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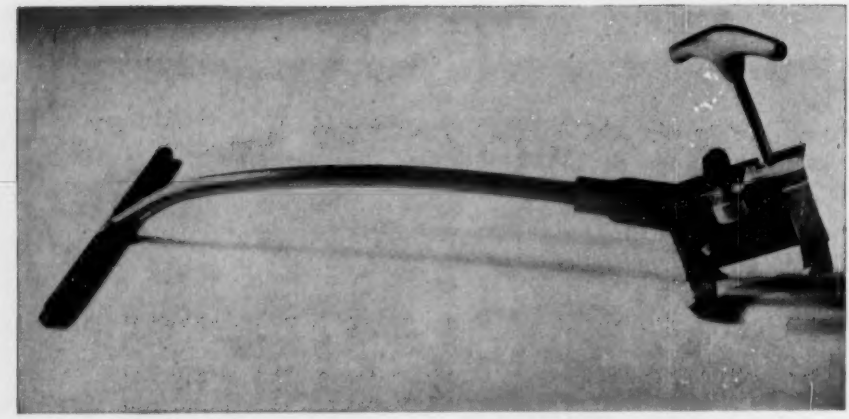

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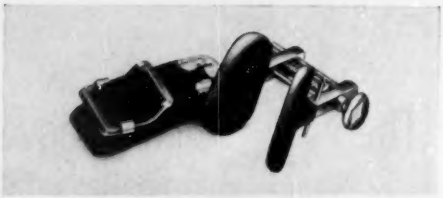

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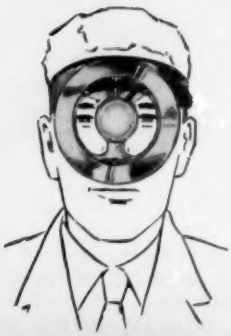

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SPONTANEOUS HEMORRHAGE

A Clinical Entity, with Special Reference to Epistaxis

PHILIP JACOBSON, M.D.
PETERSBURG, VA.

THE CHARACTER and significance of epistaxis have never been understood. In 1941 I proposed the hypothesis that spontaneous hemorrhage is a clinical entity in itself and that epistaxis is the commonest form of this disease.[1] Some time later an elaboration of this concept, with a report of 49 patients who had 73 bleeding episodes, was published.[2] Since then this study has continued and broadened into other fields, but no similar experience appeared until 1952, when Koch and his associates [3] reported five patients with hereditary hemorrhagic telangiectasia relieved by the same means as I had employed. Although I have had the experience of successfully treating well over 300 patients with nosebleeding by the intramuscular administration of estrogens, this contribution contains the important features of the cases of only eight patients with overwhelming epistaxis controlled by huge doses of estrogens administered intravenously. Four other cases of peculiar interest will be included. I believe that these speculations now have crystallized to the degree that this hypothesis is a theory that can be considered tenable. Its ramifications and implications have expanded to such an extent that they can be mentioned only briefly here.

REPORT OF CASES *

Only the essential details of these cases will be related. Such factors as the red cell, white cell, and platelet counts will be omitted, because they have been found to be uninformative. The coagulability of the blood plays no part, since in all instances it was normal. In fact, clotting often interferred with removal of blood from the nostrils. Blood pressure is not a component except in so far as hypertension enhances the rate of flow, although there have been instances of equally voluminous bleeding in persons with moderate tension. All these patients would have been candidates for extensive and repeated packings or difficult cauterizations and, according to my previous experience, would have bled right through the packs for days. Thus the suffering, distress, and inconvenience attending the use of these devices happily were averted.

CASE 1.—H. F., a Negro, aged 40, was injured in an automobile accident. He sustained some bruises about the face and a few small lacerations, which were sutured and healed promptly. Ten days later he suddenly began bleeding from the nose. The blood flowed freely and was escaping in such large quantities from both nares that it was impossible to see from where it was

Ayerst, McKenna & Harrison, Ltd., New York, generously supplied the conjugated estrogenic substance (Intravenous I remarin) used in the treatment of these cases.

* Nine of these 12 cases were treated by Dr. Julius Hopkins, of Petersburg, Va., Diplomate of the American Board of Otolaryngology. Since this article was written six more patients with overwhelming epistaxis have been treated, with the same results.

coming. Twenty milligrams of estrogen was given intravenously, and he was placed on a cot. The flow continued for about 35 minutes, and during this time it is estimated between 500 and 600 cc. was lost. There was no alarm over this large loss of blood, since I have found that these patients can tolerate a much greater blood loss than normal persons. Suddenly he began to complain of nausea and headache, which were recognized as the toxic effects of estrogen, and abruptly and dramatically the bleeding stopped, almost as one turns off a faucet. The bleeding did not recur, and when the nose was examined the next day, no prominent blood vessels were present. The toxic effects of estrogen were exactly the same as those occurring in women who have been given excessive doses of diethylstilbestrol.

Case 2.—Mrs. H. L. C., aged 50, had had infrequent moderate nosebleeding since the menopause had been completed, about five years previously. During the middle of the day epistaxis began and gradually increased in volume until she was seen about five hours later. Blood was flowing from both nares, and some was coming from her mouth, evidently rolling down into the nasopharynx. She was given 20 mg. of estrogen intravenously, and the bleeding stopped within 25 minutes, but no toxic symptoms occurred. After a while she was sent home, but a few hours later, in the middle of the night, she returned with blood flowing only from the right naris. Another 20 mg. was injected, and the bleeding stopped in about 15 minutes. There was no evidence of estrogen toxemia, and two days later only a few small vessels could be seen on either side of the septum. She has had no recurrence in eight months.

Case 3.—C. L. W., a white man, aged 65, awoke, after having been asleep only an hour, to find that his nose was bleeding; he was brought to the hospital. The left naris was full of clots, and blood was flowing furiously around them. When the clots were removed, a steady, fast stream was running down the floor of the nostril from a point midway between the angle of the septum and the floor. The actual bleeding point was obscured by a deep deflected cleft. Twenty milligrams of estrogen was given intravenously, and the bleeding stopped in 25 minutes. He was sent home but returned in two hours because the bleeding had resumed. He was admitted to the hospital and 20 mg. more administered. Seven hours later another mild episode occurred, which ceased in a few minutes without treatment. He was discharged 48 hours after admission.

Case 4.—B. G., a Negro, aged 25, had violent, sudden epistaxis while sitting on his front porch. The bleeding was so profuse that he had to hold his head forward and chin down to prevent the large volume of blood flowing into the pharynx from choking him. Blood clots were removed from the left nostril, and the bleeding point was observed high up on the dorsal surface of the septum. Thirty minutes after 20 mg. of estrogen had been given intravenously the bleeding stopped and did not recur.

Case 5.—M. W., a white man, aged 34, was awakened from sleep with epistaxis. The blood was pouring out of his right naris, but the bleeding area was obscured by a wide deviation of the septum. He was admitted to the hospital, and 20 mg. of estrogen was given intravenously. The bleeding stopped in 20 minutes. He had two more moderately profuse episodes, for which another 20 mg. was administered, and was discharged after four days.

Case 6.—L. M. T., a white woman, aged 65, who had suffered from hypertension for years, was admitted to the hospital with severe epistaxis. The blood pressure was 240 systolic and 140 diastolic. The bleeding stopped in 30 minutes after the intravenous administration of estrogen, but the drug was continued orally every four hours. There were several short periods of bleeding during the first 24 hours of her stay, and she was sent home four days later. Within 12 hours she returned bleeding profusely, but after another intravenous injection of estrogen the epistaxis ceased in 15 minutes, and she was discharged 48 hours later. The blood pressure had declined to 200/130.

Case 7.—L. C., a white woman, aged 54, had many attacks of epistaxis, often lasting for days, during the past six years. The bleeding always came from Kiesselbach's area on either side of the nose. This attack, which was more intense than usual, was easily controlled with intravenous estrogen, but ordinarily she could be relieved by oral or intramuscular estrogens.

Case 8.—A. M. H., a Negro, aged 28, began bleeding severely from his nose while at dinner. He was brought to the hospital, where he was given 20 mg. of estrogen intravenously. The bleeding ceased in 30 minutes, and a short time later he was sent home, only to return in three hours and was admitted. The bleeding was arrested again after another 20 mg. was given. He stayed in the hospital two days but had no further recurrence.

Of the four other patients of special interest to rhinologists, three were pregnant women, but only oral and intramuscular estrogens were employed. One patient had similar experiences in two successive pregnancies two years apart. Epistaxis began during the fifth month of pregnancy, and on both occasions the bleeding quickly subsided and did not recur after she received oral estrogen. The second patient during the fourth month had intermittent epistaxis, which was easily controlled the same way. The third woman, during her second pregnancy, began bleeding from her nose during the third month. The bleeding continued sporadically for three weeks despite repeated packings and cauterizations. After intramuscular administration of estrogen, the bleeding subsided. Two more injections were given during the next week. The pregnancy was completed without further mishap.

The fourth patient was a white boy, aged 15 years, who was brought to the hospital with severe hemoptysis. The nose and lungs were clear, and the blood seemed to be welling up from his throat, where the bleeding point could be seen with a laryngeal mirror. It consisted of a bluish pulsating area about 5 mm. in diameter on the base of the tongue, from which blood was streaming down into the hypopharynx. Twenty minutes after the intramuscular injection of estrogen, the hemorrhage was arrested, and the bleeding area had shrunk to less than one-third its original dimensions. During his hospital stay of three days he did not have a recurrence. After another similar episode an attempt was made to destroy this vascular area with a sclerosing solution. The only results were considerable discomfort and complete failure. He has had five more attacks during the last two years. The bleeding subsides rapidly, and the vessels almost disappear after estrogen has been given. The ultimate outcome of a succession of hemorrhages such as this patient has endured is the growth and development of a permanent area of telangiectasis. But, even if this area could be eliminated by either excision, coagulation, or cauterization, he will, since he still has the disease, establish another focus for bleeding. And, more than likely, it may be in a much more sinister location.

DEFINITION OF THE DISEASE

Sudden, unexpected, and sometimes unsuspected hemorrhage is an extremely common phenomenon in man. Such bleeding in its milder forms is so general that there are few indeed who have escaped this experience during their lifetime. The commonest varieties, such as epistaxis in children or the rectal and uterine bleeding of middle age, are relatively benign. But in the latter years, blood frequently is released abruptly into an organ, cavity, or space, where its presence can have a calamitous portent or be the precursor of a catastrophe. This bleeding is of quite another sort, with its malignity augmented by inaccessibility and not uncommonly by the mystery of its location or even of its existence. If the hemorrhagic state is inevitable, one is fortunate indeed to have the focus of bleeding in the nose. The word "spontaneous" is applied to this type of bleeding, with the full knowledge that no hemorrhage just happens without provocation or is, according to the once-prevalent absurd notion, a means of reducing tension or disposing of excess blood. The term is not accurate and should be considered a misnomer, but it is particularly apropos in that it conveys the impression of sudden, voluminous bleeding without preliminary warning.

According to this definition then, a hemorrhage, to be considered spontaneous, must happen without a reason or cause—a fallacious assumption. So far, the search for more tangible origins has led to the discovery of damaged vessels and bleeding ulcers, but even these cannot always be found. In the brain and subarachnoid space, the blame is usually put upon small sacculations or aneurysms. That these dilatations are congenital is doubtful, but there is still no explanation of why they rup-

ture, especially during and after stress. Another common form is the subintimal minute hemorrhages in coronary arteries that precede thrombosis and occlusion of these vessels.† Hemorrhage of obscure origin also takes place in the gastrointestinal and urogenital tracts, in the eye as well as beneath the conjunctiva, in the pancreas, the bronchi, the peritoneal cavity, and elsewhere. A broad and unique experience with estrogens in all of these conditions warrants the opinion that they are not dissimilar. Careful and complete examinations to establish a diagnosis always are made. When a cause for bleeding, such as tumors, cirrhosis, circulatory stasis, blood dyscrasias, trauma, infection, or any other cause, cannot be found, the hemorrhage is considered, for practical purposes, to be spontaneous. However, the source of bleeding and the cause for bleeding must be carefully differentiated. Lesions like ulcers in the stomach and duodenum or ruptured varices around the esophagus or the vesical neck may be the source but not the true cause of hemorrhage.

The analogy between epistaxis and bleeding from peptic ulcers, aside from their reactions to estrogens, may be drawn from the facts that the two have comparable psychic components and the same tendency to recur, their cause is unknown, and their response to conventional therapy has been equally uncertain and disappointing. Jordan [11] states:

> Hemorrhage in peptic ulcer is a complication which, as time and ulcer experience go on, often has been found to test the clinical acumen and judgment of both gastroenterologist and surgeon. The diagnosis of ulcer in gastrointestinal bleeding is often difficult to establish, even with surgical exploration. . . . Ulcer bleeders have a deplorable tendency to bleed after all forms of treatment, medical and surgical, . . . for statistics are gradually accumulating which indicate a considerable percentage of hemorrhage following even very high resections.

Lewison,[12] in an exhaustive review of bleeding peptic ulcer, begins his discussion upon a note of pessimism:

> The treatment of bleeding peptic ulcer has been the subject of a free and voluminous discussion in the contemporary as well as the time-honored annals of medical literature. If a final keystone to this important and perplexing therapeutic problem has not as yet been found, it most certainly cannot be attributed to medical or surgical inertness. The present prolixity and immense fecundity of the current press indicates an eager interest in the problem of peptic ulcer and its major perilous complication, namely, hemorrhage.

After observing the effects of repeated impulses from psychic disturbances and reflex stimuli from the ulcer and noting that hemorrhage can occur from penetration into major arteries, he goes on to point out that, on the other hand, profuse bleeding can come from an area of healing granulation tissue and that the extent and severity of bleeding may not necessarily be related to the size of the ulcer or the magnitude of the vessel from which blood is escaping. In fact, Lewison claims that many bleeding ulcers are not due to actual penetration into a large blood vessel and that most frequently the lateral wall of a vessel which may have undergone some degree of weakening and aneurysmal dilation is the source of hemorrhage. He ends his review by emphasizing the enigmatic nature of this bleeding with a remarkable conclusion that is just as applicable to epistaxis:

> In the halcyon years ahead, those men of medicine who may perchance read these pages should do so in the full realization of how dense and baffling was our present veil of the unknown. Our current concept of treatment for bleeding peptic ulcer constitutes merely a single facet, albeit

† References 7 to 10.

a most important one, in the medical, surgical, and economic aspects of a broader problem. . . . Perhaps the compassion and wisdom of future experience will forgive our present clumsiness, and in the full light of tomorrow we shall be able to look forward to new and more suitable drugs which will enable us to conquer the stark sequelae of vagotonia and ulcer. However, until such time as a sovereign remedy to brave the bastion of bleeding ulcer appears, our therapeutic armamentarium must rest upon present devices. Final judgment is forced upon us all by clinical experience alone. And to quote Shakespeare, "How this audit stands, no one knows, save Heaven."

The theory of fluctuating hormones, from whatever origin, which may explain these hemorrhages, will be considered after discussing some aspects of the nature of epistaxis. If this theory of "spontaneous" bleeding finally prevails, then what is now considered to be many diseases actually may be a single disease with a multitude of manifestations, depending where it strikes, and has a greater morbidity and mortality than any other human affliction.

NATURE OF EPISTAXIS

I am well aware of the fact that in the majority of patients, especially children, nosebleed stops of its own accord. But, if a sequence of events or a succession of constant effects occur with striking regularity, the results no longer can be attributed to coincidence or accident. Favorable response to estrogen therapy has been elicited too many times to dismiss this good fortune as a fortuitous circumstance. Moreover, it prompts a closer examination of some of the aspects of epistaxis that, to say the least, are perplexing.

Mention already has been made that nosebleed often stops of its own accord. But why it starts and why it stops remain unanswered. There is normally no vessel on the nasal septum capable of emitting so much blood as is so frequently encountered. How does such a large vessel or such large vessels come into being? When the bleeding stops, these vessels disappear, and what a few hours before was the scene of turbulent hemorrhagic activity subsides into a calm and placid surface with hardly a trace of its former ferment. What is the inherent peculiarity which renders ineffective ordinary measures against this hemorrhage and seems almost to force blood right through or around any barrier? Why does the blood flow in such volume sometimes as to constitute a serious hemorrhage? Is the nose just the commonest site for this phenomenon, or does it occur anywhere else? Why do other symptoms not related to hemorrhage accompany the bleeding?

Unless one chooses to deny their validity, these questions present problems that as yet are unsolved. To me, the solution lies somewhere in the realm of hormone imbalances, particularly estrogen deprivation, but that does not exclude by any means other hormones and other factors. When further intrinsic components and elements, such as basic personality, emotional upheavals, environmental situations, and heredity, are interjected, the integration of all these constituents leads into a labyrinth of conjecture that is bewildering and too far-flung to be undertaken here.

THE HORMONE THEORY

Periodic variations in the estrogen content of the blood are constant in normal women. At definite intervals the estrogen level declines and is followed by the shedding of the endometrium and bleeding. This separation takes place where the spiral arteries of the uterus enter the endometrium from the myometrium. At this

point vessels rupture, and the menstrual flow begins. The reason that the vessels rupture has been ascribed by Smith and Smith to the elaboration of a vascular toxin which accompanies any reduction of estrogen.‡ They contend that this substance is a euglobulin-like material, which they discovered in the menstrual discharge. The pathological effect of this material they consider to be of generalized vascular damage, and it may be logically supposed to be the bleeding factor, the precipitating cause of the flow. Withdrawal of estrogenic stimulation is all that appears to be needed to result in tissue catabolism and the concomitant formation of a toxin that has a peculiar affinity for the vascular system. Since men as well as women have estrogens circulating in their blood, it is not unreasonable to assume that conditions and circumstances can arise which will depress the level of this hormone in either sex. When this depression continues, the toxin begins its attack on the vascular system. Thus the human organism contains a bleeding mechanism that needs only suitable and adequate stimuli to provoke it into operation, an unhappy experience that is only too familiar.

The effects of this toxin can be seen occasionally in women with varicose veins. Just before and during menstruation these veins will enlarge, become tender and painful, and present the appearance almost of being inflamed. Sometimes this chemical inflammatory process will persist until thrombi are induced; and phlebothrombosis certainly is not unusual in either sex. If it is assumed then that after a period of estrogen reduction the toxin is produced in increasing quantities, it may affect the vessels anywhere, weakening their walls, dilating their lumens, and increasing their fragility until eventually an actual rupture is precipitated. The focal point for bleeding, which usually consists of one large and several smaller vessels, is thus prepared. But why any one site is singled out is a mystery. My experience with the use of estrogen for spontaneous hemorrhage precedes the publication of Smith and Smith's work and was carried out on an empirical basis without any real knowledge of the mechanisms involved. This study was prompted by the observation that reduction in blood estrogens, manifested by so-called hot flashes, often was associated with bleeding from the nose, stomach, and elsewher in women beyond the menopause. Many of these women had no endometrium, since it had been removed by hysterectomy or destroyed by radium when that vicious treatment for functional bleeding was in its heyday.

THERAPY OF EPISTAXIS

Careful examination of the patient will disclose that, while epistaxis is the prominent and presenting sign, there are other characters which should not be overlooked. The pulse is full, bounding, rapid, and forceful, but this sign cannot be interpreted as being the result of hemorrhage, since when the bleeding ceases the pulse will return to normal despite the loss of blood and without replacement. This return often occurs before the stream of blood begins to dwindle and can be used as a guide in the treatment. The patient is agitated and in the obvious alarm and panic that is to be expected. But many bleeding episodes happen, just as do so many other cardiovascular accidents, in the middle of the night when the patients are supposedly fast asleep, and many persons have recounted that before awakening they had a bad dream or were excited and tense even before the bleeding was discovered.

‡ References 4 to 6.

This increase in psychic tension is an important sign and its reduction one of the objectives of treatment. Sedatives, assurance, and a calm and confident demeanor are essential for managing this aspect of the disease.

At first I was unaware of the quantities of estrogen the human organism could tolerate clinically in this condition, and I used the substance cautiously and carefully. One milligram repeated in four to six hours was the average dose for adults and 0.5 mg. for children. It is interesting to note that this small quantity was given to several children with delayed post-tonsillectomy bleeding, with gratifying results. It did not take long to learn that much larger quantities produced no ill effects, but the idea of increasing the dose up to the point of toxemia did not occur to me. In patient after patient the same results were achieved. In 30 to 40 minutes after the intramuscular injection was given and without any treatment to the nose itself, the flow of blood diminished and eventually ceased altogether. Sometimes it would recur a few hours later, and another injection would be given. The quantity of blood lost did not affect the procedure, since, as already stated, these patients can withstand the loss of large quantities of blood without ill effects. An incidental finding was that these people do not take transfusion too well and that the number of transfusions must be limited, if indeed, they are needed at all.

It became apparent during the therapy of other conditions, notably endometriosis, that much larger doses were tolerated with ease. The availability in January, 1952, of estrogen compounds to be injected intravenously for functional uterine hemorrhage made the task much simpler. Here was a drug that could be given to the point of toxemia without ill effects other than minor symptoms, and if it did no good at least could do no harm. Accordingly, I have not hesitated to employ this material in the manner described. Moreover, it can be used almost indiscriminately, since, if a definite reason for the bleeding should be discovered, no injury can ensue.

The large discrepancies in the dosage requirements are puzzling and may depend on variables in the rate of absorption, conversion, and destruction of estrogens in different persons. Moreover, the effects of estrogens do not appear gradually. There seems to be an interval between injection and action during which nothing happens. Apparently a period of time is necessary for the utilization of the hormone before any results are perceptible. This study has been limited to clinical observation, since neither standards for the normal values and variations of the circulating sex hormones have been determined, nor have quantitative methods simple enough for general use been invented. These substances cannot be measured with the rapidity and accuracy of sugar, nitrogen, or other constituents of the blood stream. With this disadvantage, only one pertinent fact has been fairly well established: The quantity of excreted 17-ketosteroids, which are related compounds, declines with age. Hence, at present there is no way of detecting and preventing an impending bleeding episode by examination of the blood.

SUMMARY

I am of the opinion, so far supported only by clinical observation, that episodes of spontaneous hemorrhage, which include epistaxis, are not isolated incidents in themselves but are the manifestation of a systemic disease of which bleeding is the principal sign. They arise out of a reduction in the circulating blood estrogens, and this deficiency incites the formation of a tissue toxin that promotes vascular catabolism, especially of the veins. The cause of this decline is unknown, but it may be

due to involved emotional complexes similar to those which, as various authors have clearly demonstrated, gravely affect the entire endocrine pattern. The role of heredity also is unknown. Hence, spontaneous hemorrhage, wherever it may occur, is a clinical entity in itself and may yet be regarded as one of the diseases of the general adaptation syndrome of Selye.

Epistaxis, the commonest form of spontaneous hemorrhage, can be arrested by adequate and, if necessary, frequent doses of estrogen without the necessity for any physical agency, such as packs. Eight cases of overwhelming epistaxis and four other cases of nosebleed, all controlled by the oral or parenteral administration of estrogen, are presented. The rationale of this therapy rests upon the immediate correction of the state of estrogen deprivation and rejection of the popular impression that mechanical arrest of the hemorrhage constitutes a cure of the disease.

I have no intention of insisting upon estrogen as a panacea for spontaneous bleeding. Besides, it can be of little benefit after serious damage has been inflicted. Other hormones and compounds and other agents yet to be discovered may prove more efficacious. The purpose of this communication is to advance the conception that epistaxis is a systemic disease and differs from similar hemorrhages of obscure origin only by virtue of the fact that the bleeding does not take place in a vital or confined location.

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PROBLEMS IN VASOMOTOR RHINITIS

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THE TERM vasomotor rhinitis is a confusing one. It was originally used to include all those symptoms which are now described as due to nasal allergy. The commonest of these, of course, are itching, paroxysmal sneezing, profuse watery nasal discharge, uniform swelling of the turbinate bodies, grayish white mucosa, and eosinophilia. When these are found in persons who have positive skin-test reactions to the common pollens, the more modern term is "pollinosis." If patients show positive reactions to the routine offending contactants, we now refer to them as suffering from specific "contactant rhinitis" or "contactant allergy."

In this discussion, however, we shall consider under the term "vasomotor rhinitis" that very large group of patients whose primary symptoms are those mentioned above and yet who show no positive skin reactions following either scratch or intradermal testing. It has been suggested by Hansel [1] that their complaints may be due to increased mucous membrane sensitivity with no alteration in the sensitivity of the skin. This seems to logically explain many of the complaints, and yet there must be other underlying causes.

Probably the largest number of these patients are annoyed by the sudden onset of their symptoms following a change in atmospheric temperature or humidity or following eating. Moving in or out of an air-conditioned building or theater will often initiate sneezing or rhinorrhea that may last for hours. Some may have their symptoms aggravated for a whole summer while encountering high atmospheric humidity. All are uncomfortable and need assistance.

The rhinoscopic examination of these patients usually shows the typical pale, boggy mucosa, but mucopus is rarely present and there is little or no tenderness over the sinus areas. Much watery discharge is present, and the inferior turbinates are universally hypertrophied from the vestibule to the nasopharynx. Polypi are not usually present. Nasopharyngoscopy or posterior rhinoscopy shows the posterior tips of the inferior turbinates to be "mulberry" in type and of a purple color.

The most important single finding in the diagnosis of these cases, however, is the presence of eosinophiles on cytologic examination of the nasal secretions. Such examinations should be done routinely in all suspected cases.[2] The technique is

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simple and requires little time and space. The specimen is taken by first having the patient blow his nose in a small piece of waxed paper. A fleck of the mucoid secretion is then spread on a glass slide, dried, and covered with either Hansel's or Wright's stain. After this, it is diluted with water and washed with alcohol and again dried. The microscopic examination is made with both high-power and oil-immersion lenses, and the number of eosinophiles and neutrophiles is noted. Eosinophiles in qauntity and alone indicate uncomplicated allergy or vasomotor rhinitis. Neutrophiles in large numbers indicate infection. A combination of the two types of cells denotes a complicated case in which both allergy or vasomotor rhinitis and infection coexist. The type of cells predominating indicates which condition is the more important one.

The exact role of the eosinophile in allergy is unknown, but it has been suggested that it is concerned with the disposition or elimination of foreign or broken-down protein in the blood and tissue. Code [8] has shown that the eosinophile is a carrier of histamine, but it is not known whether it carries this substance to the tissues or away from them.

Intradermal tests with histamine diphosphate in 1:100 and 1:1,000 dilutions will frequently give a marked reaction. Approximately 0.05 cc. of the test dilutions are injected into the skin about 2 in. (5 cm.) apart, and they are compared with similar opposing injections of isotonic saline solution or sterile water. In patients suffering from vasomotor rhinitis there is usually an increased histamine reaction.

TREATMENT

The treatment of these cases is both surgical and medical and both temporary and permanent. A combination of all four is usually necessary to produce effective therapeutic results.

On the patient's first visit, the nasal cavities, turbinates, meatuses, and mucosa of both the nose and the nasopharynx should be carefully investigated without shrinkage. Next the same careful examination should be made after the interior of the nose has been sprayed with a standard shrinking agent in isotonic solution. If polypi are found and are obstructive to comfortable breathing or to adequate sinus aeration and drainage, they should be removed surgically. This is best accomplished with the Krause snare under topical anesthesia of either cocaine or tetracaine (Pontocaine) hydrochloride. Small stubs that remain after the polypi have been snared should be removed with a Gruenwald punch. One should always remember the nasopharynx when doing a polypectomy and inspect it very carefully, for it is uncommon to find multiple polypi in the nasal passages without similar and often larger growths in the nasopharynx.

If the nasal septum is truly deviated or if spurs are present which block either the airway or the meatuses, a submucous resection should be performed to give satisfactory permanent comfort.

When the inferior turbinate bodies are hypertrophied and remain large after shrinkage, it signifies that the underlying bone is also large and should be removed. This can be done effectively by submucous resection of one or both of the inferior turbinate bones, with subsequent retention of the mucosa intact. It is performed by

anesthetizing the anterior one-third of the turbinate body, incising the mucosa and mucoperiosteum from behind forward, and separating it from the underlying bone, both superiorly and inferiorly. The bone is then removed from its attachment, and the subsequent reduction in size of the turbinate is permanent. In a series of 127 cases operated on bilaterally in this manner, House [4] found the procedure to be very satisfactory. His observations and conclusions were made two to four years after the surgery had been performed.

If the inferior turbinates become small after thorough shrinkage, then it may be concluded that the underlying bone is also small and the enlargement due to soft tissue swelling. Here, electrocoagulation of the engorged tissue is a very gratifying procedure. This is probably the most frequently performed surgical procedure in the treatment of vasomotor rhinitis, and it is one of the most successful. It should be done under both topical and infiltration anesthesia by drawing the electrodes anteriorly along the surface of the turbinate body from the nasopharynx to the anterior tip. Only one side should be treated at a visit, and five to seven days should intervene before the opposite turbinate is treated. This prevents undue discomfort from a temporary increase in blockage due to edema and crust formation. The treatment may be repeated once or twice to remove all tissue which is obstructive. Electrocoagulation, when used in a patient suffering from turbinate hypertrophy due to vasomotor rhinitis, gives excellent permanent results which are greatly appreciated.

Probably the most gratifying recent advances in the therapy of vasomotor rhinitis have been in the field of histamine desensitization and nonspecific antigen therapy. I say nonspecific, because many of these patients are greatly helped by fortified house-dust injections given subcutaneously or intradermally, even though they have skin-test negative reactions to repeated house-dust scratch and intradermal tests. The injections are given twice a week, commencing with 0.1 cc. of the 1:100,000 dilution. The dose is increased 0.1 or 0.2 cc. each visit until 1 cc. is reached, and then the same dosage schedule is repeated with the 1:10,000 dilution. This injection program is again repeated with the 1:1,000, 1:100, and 1:10 dilutions until definite lasting improvement is noticed. When this is reached, no further increase in dosage or dilution is necessary. Only the time interval between injections of this "maintenance" dose is altered.

Histamine diphosphate may be used in either the standard treatment method or the newer and perhaps more effective small or optimum dose therapy. The former consists of subcutaneous injections of increasing doses of the standard 2.75 mg. of histamine diphosphate in 5 cc. of diluent. One-tenth cubic centimeter of this solution is the usual initial dose. It is injected subcutaneously, and subsequent doses are increased 0.1 cc. each until the symptoms are relieved. The injections are again given twice a week and are not increased in amount after relief has been obtained. Only the frequency of the dose is then altered.

In the optimum dosage method,[5] the patient receives injections of histamine diphosphate varying in concentration from 1:100,000 to 1:10,000,000.

The strength of the dose given depends on a careful intradermal titration in which 0.5 to 0.1 cc. of each strength is introduced just below the outer layer of the

skin. These test injections are placed at about 1 in. (2.5 cm.) intervals along the inner surface of the forearm. The dilution that produces a wheal varying from 17 to 19 mm. in diameter is the one that is selected for therapy. One-tenth cubic centimeter of this strength is given subcutaneously as the initial injection, and the amount is increased 0.1 cc. each subsequent visit until satisfactory relief is obtained. When this has been reached, the dose is no further altered in amount, and it is repeated when there is cessation of comfort. This may be at the end of five to seven days, or it may not be necessary for three to four weeks. The exact physiologic mechanism of this response has not yet been satisfactorily presented, but it has been observed so many times that it must be remembered when one is considering modern therapy in vasomotor rhinitis.

While waiting for the full therapeutic effect of either regular or optimum-dosage immune therapy, I can see no reason for not using antihistaminic drugs to relieve the annoying nasal blockage, rhinorrhea, and sneezing. These may be used in tablet form three or four times a day. They should, however, be gradually withdrawn as the immunologic therapy becomes more effective. The same may be said for bland isotonic vasoconstricting nasal sprays. They will frequently give immense temporary comfort to the patient without exerting any permanent harm to the mucosa. They, too, should be withdrawn when local therapy is not necessary for a comfortable and adequate nasal airway.

SUMMARY AND CONCLUSIONS

These then are the steps to be taken in the diagnosis and effective surgical and medical treatment of vasomotor rhinitis:

1. The nose, nasal pharynx, and oral pharynx should be carefully investigated, and the color of the mucosa, the amount and type of secretion, the condition of the turbinates, and the presence of polypi should be noted.

2. Cytologic examination with Hansel's or Wright's stain should be done on all questionable cases and the number of eosinophiles and neutrophiles noted.

3. If polypi are present in any nasal passage and are obstructive, they should be removed surgically.

4. If the septum is markedly deviated, with or without spurs, and produces obstruction to satisfactory nasal breathing, a submucous resection should be performed.

5. Hypertrophic inferior turbinates which produce blockage of the nasal passages should be electrocoagulated under local anesthesia, or a submucous resection of the anterior tips should be performed to reestablish comfortable breathing.

6. The patient should receive immunizing doses of fortified house dust subcutaneously, along with similar immunizing doses of histamine diphosphate. The therapy is more effective and lasts for a longer period of time when the optimum dosage method is used after skin titration for the selection of the proper d:: :tion.

7. There is no objection to using bland isotonic shrinking sprays and antihistaminic medication orally while establishing immunity.

Although a great deal of research is still to be done in this interesting field, our patients can be made more comfortable if these principles are considered in outlining their diagnosis and treatment.

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EMERGENCY TREATMENT OF BLEEDING ESOPHAGEAL VARICES

Treatment by Transesophagoscopic Sclerosing Injection Plus Pneumatic Tamponade

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THE CRITICAL problem presented by the cirrhotic patient during massive hemorrhage from esophageal varices can be attacked actively by either the transthoracic or the peroral route. An entirely satisfactory way to stop variceal hemorrhage has not yet been found, and all must agree that at times it is impossible to control it by any means. Control is urgent, not only because of the blood loss itself but also for prevention of liver failure. If blood is readily available for transfusion, liver failure is more frequently a threat to life than exsanguination. Therefore it is most important that hemorrhage be checked quickly, even though blood replacement can be kept abreast of blood loss.

The goal of emergency treatment of bleeding esophageal varices at Walter Reed Army Hospital is control of bleeding until portal decompression—preferably by portacaval shunt—can be accomplished. It is believed that at present no definitive procedure can safely be carried out during active bleeding. Partial esophagogastrectomy, proposed as a maneuver which combines both emergency and definitive treatment, has the objection of increasing portal hypertension and thus encouraging development of gastric varices and erosive gastritis. Local experience with two instances of portacaval shunt performed during active hemorrhage was not encouraging. Thoracotomy with transmural varix ligation has been reported as effective in certain cases but has relative disadvantages of delay and danger secondary to the mechanics of major surgery.

Transesophagoscopic sclerosing injection of varices which are not bleeding, as an elective and definitive form of treatment, has received support from several quarters.* The results have been moderately encouraging, although development of the shunting techniques has now limited its application to the unusual clinical situation. There will probably always be a place for sclerosing therapy, and an attempt to increase the effectiveness of the technique is a worth-while line of endeavor. The extensive literature on elective sclerosis includes a disturbing note, in that some investigators have been unable to find histopathologically in autopsy specimens any evidence of varix thrombosis or inflammatory reaction following injection. Initiation of sclerosis must depend to a large extent on intravariceal retention of the sclerosing agent plus a degree of active assistance in apposition of

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* References 1 and 2.

the varix walls. It was thought that addition to the technique of induced venous stasis prior to injection, by pressure exerted at the cardia, and compression of the varices immediately following deposition of the sclerosing agent by pneumatic tamponade might render varix obliteration more effective.

One of the advantages at times realized from the vigorous diagnostic approach to massive upper gastrointestinal hemorrhage [3] is detection of a bleeding lesion which can be treated on the spot. There are reported in this article experiences in the emergency therapy of bleeding esophageal varices by transesophagoscopic sclerosis and tamponade at the time the varices were first detected, by esophagoscopy. A group handled in the same way, with the exception that no sclerosing injection was made, will be discussed briefly as an imperfect control series. All patients were hospitalized because of massive hematemesis, and, although some had bled previously and some upon initial examination showed the stigmata of cirrhosis, in none had any potentially bleeding lesion ever been found or was the source of the current hemorrhage known.

MATERIAL

Twenty-five patients, aged 22 to 60 years and proved histopathologically to have portal cirrhosis, were treated. Seventeen formed the control group and will be discussed later. Among the other eight, who were treated by injection in addition to tamponade, there were four who were hospitalized within an hour or two of onset of hematemesis. The rest were transferred from other medical facilities during active bleeding, which had begun from 14 to 48 hours previously. The latter patients had already received from 1,500 to 5,000 ml. of whole blood. Each of the eight patients was in desperate condition at the time of manipulation, and, in spite of continuous transfusions into more than one extremity before and during esophagoscopy, four were in a state of clinical shock at the time. One patient was comatose from liver failure, and altogether seven had liver failure within four days of the time of manipulation.

TECHNIQUES

Although, of course, individual patients were treated according to the particular problems involved, a plan of procedure has become rather well stabilized, because under the desperate conditions of massive hematemesis there can be no uncertainty of action. The following routine was used in the present study.

Immediately upon admission, the history and physical examination are completed and necessary blood transfusions arranged for and started while the patient, still dressed, is on the stretcher. Meanwhile, a 30 F. stomach tube is passed and the stomach lavaged with ice water. The success of the whole effort depends largely on thoroughness of lavage, and 20 minutes may be required, even though a large tube is used. A narrow tube is not at all effective. Bleeding from esophageal varices frequently ceases during ice-water lavage, and this in itself is a good form of momentary treatment.

Esophagoscopy under 4-diameter telescopic magnification follows, on the stretcher, on the patient's bed, or wherever convenient. If the information thus far gathered has suggested the possibility of cirrhosis, a Sengstaken tube is passed into the stomach before esophagoscopy, the gastric balloon inflated, and gastric aspiration begun. If, on the other hand, varices are encountered unexpectedly, the Sengstaken tube is passed along the side of the esophagoscope. Because in this class of patient one

approaches the esophagus without information about its configuration, the esophagoscope must be passed under direct vision rather than with the help of an obturator.

One is not able to identify the bleeding point with certainty in every case, and, paradoxically, the remarkably clean state of the esophagus at times hinders location of the source. Rarely, blood is found to be issuing from a long segment of a varix. It is important that the varix selected for sclerosis be injected as far distally as possible, preferably just above the esophagogastric junction, even though the bleeding point lies several centimeters proximally. Parenthetically, because of the possibility of aggravating hemorrhage elsewhere in the stomach, it may be a mistake to inject bleeding gastric varices; this is an unexplored technique, however, and no positive opinion can be expressed. The presence of gastric varices—a state of affairs which can be determined only by gastroscopy—is a contraindication to elective sclerosis of esophageal varices, but during massive hemorrhage this consideration is perhaps best ignored.

There is something to be gained for later purposes by taking the opportunity to measure the portal vein pressure just before the sclerosing solution is injected. If the first attempt is not successful, however, the exigencies of the more important problem usually preclude further efforts. The gastric balloon is drawn up against the cardia, and 3 ml. of 5% sodium morrhuate, plus the amount necessary to fill shaft and connections of the needle, is injected into the varix lumen. It has been the practice then to inject immediately a similar amount into each of two nearby varices, chosen at random. The esophagoscope is then withdrawn and the esophageal balloon immediately inflated. Because it is possible that success of the venture depends in part on how quickly the varices are compressed after deposition of the sodium morrhuate, the former practice of packing the region of the bleeding site with oxidized cellulose (Oxycel) has been discontinued. It has probably become a superfluous gesture, too, now that reliance for tamponade has been placed on the Sengstaken tube. The time lapse between the first of the three injections and inflation of the esophageal balloon amounts to about 20 seconds. The Sengstaken tube is maintained in place for at least five days, with continuous manometric control over the balloon pressures.

The means of entering the varix with the needle, of ascertaining proper needle position, and of maintaining an emotional state of relative equanimity are not learned from a written description. It must be emphasized that technically the first two are not at all difficult. It must be admitted, too, that the operator may on occasion feel rather insecure concerning the exact location of his injection. Sloughs have been reported following misplaced sodium morrhuate, but there is no available information on expected incidence.

FINDINGS AT ESOPHAGOSCOPY

The varices in the segment of the esophagus most severely involved were greater than 6 mm. in diameter in six instances and between 4 and 6 mm. in two. The entire esophagus was involved in three, two-thirds in one, one-half in two, and one-quarter in two. An attempt was made to measure the portal vein pressure at six of the manipulations, and satisfactory readings were obtained four times: 540, 430, 240, and 470 mm. of water. The bleeding site was identified with certainty in five

instances. It was diffuse over a segment measuring several centimeters along a single varix in one. The other four patients were bleeding from a small area 2 to 3 cm. proximal to the esophagogastric junction.

REPORT OF CASES

CASE 1.—Into one varix was injected 3.5 ml. of sodium morrhuate. The Sengstaken tube was removed after five days. There was no evidence of further bleeding following the injection. A satisfactory portacaval shunt was established 19 days after sclerosis. When last examined, seven months later, the patient was working full time, without further bleeding.

CASE 2.—A balloon tamponade was not used. Instead, a Levin tube was passed, and, after a sclerosing injection of 2.5 ml. at one site, oxidized cellulose was packed around the tube from the injection site to the bleeding point. The tube and pack were ejected by the patient one-half hour later. Liver failure had been apparent upon admission and progressed rapidly. Sixty hours after injection there was moderate hemorrhage, and 24 hours later the patient died. From the immediate postinjection period, when the hematocrit reading was brought to 42% by transfusions, until the time of death the patient required 1,000 ml. of additional blood. Death was due to liver failure. At autopsy only streaks of old blood were found in the intestinal tract. No gross evidence of varix thrombosis or sclerosis was found, and no residuals or effects of the injection could be identified. Histopathologic examination showed the varices of the distal esophagus to be patent.

CASE 3.—One varix was injected with 3.0 ml. of sodium morrhuate while the patient was comatose in severe liver failure. She died 60 hours later, after a progressively worsening course. The Sengstaken tube remained in place until death. There was no bleeding following sclerosis. Autopsy was refused.

CASE 4.—One varix was injected with 4.0 ml. of sodium morrhuate and the hematocrit brought to 40% by whole blood transfusions. The Sengstaken tube was removed after three days. Eight days after sclerosis there was melena, which lasted for four rather large bowel movements. Whole blood, 1,000 ml., was given, after which no more was considered necessary. Four days after injection liver failure had become evident, and the patient died after progressive failure on the 21st day, without further bleeding. At autopsy the varices were discerned with difficulty. Inspection and palpation failed to demonstrate local induration or thickening, which might have indicated a sequel of sclerosis. Histologically, the mural layers of the esophagus were normal except for the varices. There was no evidence of vascular sclerosis and no inflammatory reaction through the connective tissue to suggest that injection had been paravascular.

CASE 5.—Sodium morrhaute, 3.0 ml., was injected into one varix. The Sengstaken tube was removed after five days. There was no further bleeding following sclerosis. A successful portacaval shunt was made on the 33rd day, and there had been no further bleeding when the patient was last examined, 13 months postoperatively.

CASE 6.—Three varices were injected with a total of 8.5 ml. The Sengstaken tube was removed at the end of three days, after it had proved most unreliable. At 8 and 24 hours after injection there was regurgitation of small amounts of blood from above the esophageal balloon. There was no further bleeding. The patient died of liver failure 108 hours after injection. At autopsy there was old blood in the colon but none elsewhere. Varices were demonstrated with difficulty. Grossly, no changes could be found which could be ascribed to the injections or sequels of sclerosis. Sections were taken from the esophagus at 3 and 6 cm. above the estimated sites of injection. The varices were prominent and patent. None was sclerosed, and no inflammatory reaction was found throughout the tissue.

CASE 7.—Three varices were injected with a total of 8.5 ml. and the Sengstaken tube removed after five days. There was no further bleeding. The patient died of liver failure on the 20th day after injection. At autopsy the esophagus showed superficial erosive esophagitis and several varices. There were no thickenings or induration grossly, and multiple sections revealed no varix occlusion or tissue inflammation.

CASE 8.—Three varices were injected with a total of 8.5 ml. Five Sengstaken tubes were used, one after the other intermittently over a period of three weeks, because of structural failures and damage by the patient's teeth. There were three important hemorrhages, at 7, 14, and 25 days following injection. A total of 3,500 ml. of blood was required for replacement. During the third hemorrhage an attempt at emergency portacaval shunt was made. The portal, mesenteric, and splenic veins were found unsuitable for anastomosis, and the surgeon had to be content with splenectomy. The patient has remained free of hemorrhage for one month at the time of this writing.

EXPERIENCE WITH PATIENTS TREATED BY TAMPONADE ALONE

Seventeen other patients were admitted because of massive hematemesis of unknown origin, were found by immediate esophagoscopy to be bleeding from esophageal varices, and were treated by pneumatic tamponade without sclerosing injection. Nine died before surgical portal decompression could be done, three of exsanguination, and six of liver failure secondary to hemorrhage. Seven of the nine had significant hemorrhage between the institution of tamponade therapy and death. Four patients hemorrhaged while the Sengstaken tube was in situ but mechanically impotent.

The hemorrhage of eight patients was controlled sufficiently by pneumatic tamponade alone to permit shunt surgery. The average blood replacement required between institution of tamponade and operation was 10,090 ml. per patient.

COMMENT

Local experience has shown tamponade with the Sengstaken tube to be a good technique for emergency control of bleeding varices. It is not good enough, and, in spite of constant around-the-clock attention, there are failures due to both technical and mechanical reasons. The balloons may shift and leak, and on occasion the patient may eject the tube, inflated balloons and all. An element of danger from asphyxiation attends this complication, although a rule that the tube must be passed only orally, that scissors for quickly severing the tube and its air channels must be kept tied to the free end of the tube, and that the patient must never be left unattended has largely obviated this worry.

The principal objection to tamponade is not so much the insufficient degree of reliability as the temporary relief from bleeding afforded. There is such a wide range of individual tissue tolerance to the presence of a retained esophageal tube that it is difficult to know from patient to patient how long tamponade can safely be applied. It is the rare patient, indeed, who can be properly prepared for shunt surgery within a few days of hemorrhage. The tube must usually be removed long before surgical portal decompression can be attempted. Successful tamponade appears to have no influence over propensities toward further hemorrhage after the tube has been removed. Time after time in local experience hemorrhage in all its original severity has recurred within a few or several days of the removal of the tube.

The main disadvantage of emergency sclerosis is unreliability. Although sclerosis is well adapted to quick bedside or home-call use, its effectiveness cannot be predicted at the time of treatment. If there is no further bleeding, one cannot necessarily assume that the injection has helped. If bleeding recommences, it is difficult to know whether the same or another varix has opened.

The present experience suggested that in those patients who survived liver failure, the sclerosing injections had helped prevent or reduce the severity of further hemorrhage. This can only be a general impression, because there are so many variables from one cirrhotic patient to the next. Nevertheless, one could not help being impressed by the fact that, once the hematocrit reading had been brought to a satisfactory level at the time of emergency manipulation, further hemorrhage prior to shunt surgery had required an average of eight times as much blood replacement for those treated by tamponade alone as for those treated by sclerosis plus tamponade.

There is no explanation for absence of histopathologic residuals of the injection. Others have noted this after treatment of nonbleeding varices with the same sclerosing material. The needle guard is set 4 mm. from its tip, and the angle of puncture is oblique. It is not mechanically possible for the material to be injected paraesophageally. The operator can, on the other hand, be sure in every instance that the material has been deposited in tissue, rather than into the esophageal lumen. It is believed that failure to find necrosis and inflammatory reaction through the esophageal wall indicates that the sclerosing agent was, in truth, injected intravenously in all instances. This is a point which cannot be determined with assurance at the time of injection. Although the paucity of muscle tissue in a varix wall makes one suppose that there can be little intrinsic muscular control over varix diameter, it may be that intravascular irritation of the sclerosing material discourages portal flow through the gastroesophageal anastomotic system, with reciprocal increase in utilization of the other native portacaval anastomoses. There is some evidence that such a reciprocal mechanism exists, although its control is not understood.[4]

SUMMARY AND CONCLUSIONS

Eight patients with massive hematemesis were found esophagoscopically to be bleeding from previously undetected esophageal varices and were treated on the spot by intravariceal injection of sodium morrhuate and simultaneous pneumatic tamponade. This emergency therapy was intended to assist control of hemorrhage until surgical portal decompression could be accomplished.

Hemorrhage was satisfactorily controlled through the period of definitive surgery in three cases. The patients required an average 1,170 ml. whole blood replacement for that lost between the time of injection and operation. The five other patients died from 2½ to 21 days after sclerosing injection, before surgery could be done. All deaths were due to liver failure. None died of exsanguination. Altogether there was no significant bleeding following injection in five of the eight patients. Histologic examination of four esophagi obtained at autopsy failed to reveal evidence of varix obliteration.

A control group of 17 patients, first found to have esophageal varices by esophagoscopy at the time of hemorrhage, were treated by immediate pneumatic tamponade as the only local therapy. Eight were controlled sufficiently well to permit definitive surgery but required an average of 10,090 ml. of transfused blood to replace that lost between institution of tamponade and surgery. Nine patients died as a result of hemorrhage, three from exsanguination, and six from liver failure.

Because of the vicissitudes of varices and of hemorrhage in the cirrhotic patient, it is believed that the two groups are not comparable for deciding the usefulness of adding sclerosis to tamponade for emergency control of bleeding varices. Neverthe-

less, the present experience has suggested that, however effective tamponade may be made for immediate control, addition of sclerosis injection helps reduce the amount of blood lost during the dangerous period between the time that tamponade must be discontinued and definitive surgery can be accomplished.

The fate of injected sodium morrhuate is yet to be determined.

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EFFERENT NERVE FIBERS OF COCHLEA

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ALTHOUGH the sensory terminations of the cochlear nerve have been well known since the outstanding research of Retzius, Ramón y Cajal, and, more recently, Lorente de Nó, other systems of fibers in the auditory apparatus are far from being perfectly understood. Yet it is a problem which at present occupies many researchers, for the morphological descriptions that proceed from its investigation will result in the understanding of many points, still obscure, in the auditory physiology.

For instance, in a recent article (1952), Bocca [3] states the opinion that the spiral bundles of Corti's ganglion are composed essentially of sensory fibers whose function is to catch the spatial quality of sounds (Kobrak [10]) and admits, with other authors, the possibility of a double sensory innervation to which radial (ordinary sensation) and spiral (spatial quality of sound) fibers should correspond.

Moreover, we stated in 1951 [20] that the spiral bundles of Corti's ganglion are formed almost exclusively (98% of the fibers) by the efferent fibers originating in the medulla and passing with the nerve and through the vestibular ganglion, thus agreeing with Fernandez,[8] whose research appeared about the same time, that a double innervation exists—one sensorial, or afferent (radial system), the other, efferent (spiral system).

The two opinions, very recent and entirely opposed, concerning the role of the spiral bundles show the immediacy of such researches and their physiological interest.

INVESTIGATION

We present here topographical, morphological, and experimental proof sufficient to refute the sensory characterization (Bocca [3]) of the great majority of the fibers of the spiral system and to establish their efferent character.

The diversity of opinion on the subject is accounted for by the many difficulties encountered in the study of the nerve histology of the hard part of the temporal bone. In the past few years these researches have been made easier by the use of a new technique, a silver impregnation method (Weber *), by which, in accordance with their function, morphological and tinctorial differences in the nerve fibers are obtained (Baumann [2]). This remarkable technique enabled us to follow, in the trunk of the auditory nerve, in the modiolus, and, finally, at

From the Auditory Laboratories of the Bordeaux Faculty of Medicine (Director, Prof. Georges Portmann) and the Anatomy Institute of the Geneva Faculty of Medicine (Director, Prof. J. A. Baumann).

* References 24 and 25.

the level of their terminations, in various mammals, the nerve bundles whose morphology, organization, topography, and behavior under acoustic trauma support the hypothesis of their efferent character.

The cephalic extremity of a guinea pig embryo measuring 26 mm., five temporal bones of adult guinea pigs, five of white rats, and five of kittens, sectioned in series of 10μ, after intracardial perfusion, were prepared and examined. Twenty guinea pigs were subjected to a violent noise (137 phons) for periods varying from 2 to 1,000 hours, and their temporal bones were prepared in the same manner as the ones mentioned above.

Several kinds of fibers exist in the nerve apparatus of the inner ear. The cochlear fibers, after impregnation, are of medium caliber and of dark brown

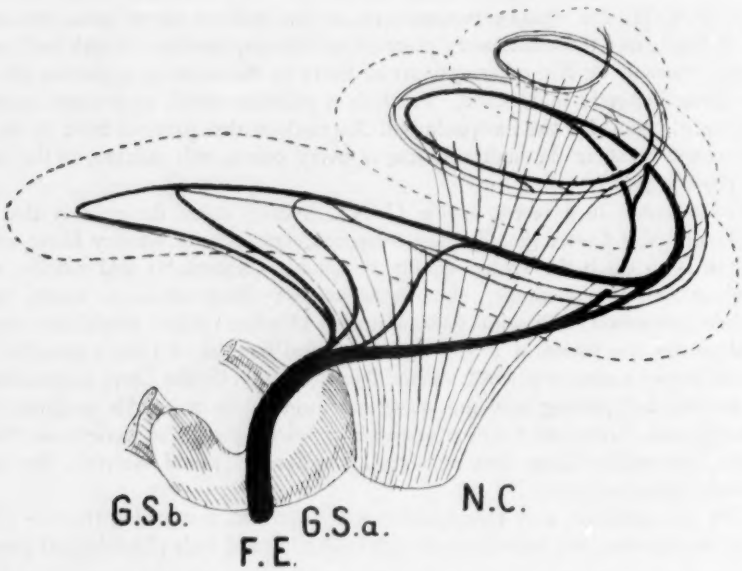

Fig. 1.—General diagram of cochlear spiral efferent bundles (in heavy black): *N.C.*, cochlear nerve; *FE.*, efferent bundle studied; *G.S.a.*, Scarpa's ganglion, saccular portion; *G.S.b.*, Scarpa's ganglion, utriculoampullar portion.

color; the vestibular fibers are on the whole somewhat thicker and lighter in color than the cochlear. There are also extremely fine fibers, very clear, terminating on the vessel walls (Kolmer,[11] Portmann and Portmann [18]) and belonging to the postganglionic portion of the thoracolumbar sympathetic (orthosympathetic) system. Finally, there exist very thin fibers, usually of a clear brown color, sometimes yellow, not so well colored as the sensory fibers, but staining more distinctly than the thoracolumbar sympathetic fibers; except at the level of their extremities, they are always grouped in bundles, in which they are tightly packed and where Schwann nuclei are encountered in large numbers. This morphological aspect being characteristic and having been observed in all sections examined, we were able to make, with relative ease, a topographical study of these efferent bundles, the precise significance of which is ignored in the present state of research.

BULBAR PORTION

The efferent cochlear bundle was well studied in the bulbar portion by Rasmussen as early as 1942.[21] The fibers arise in a nucleus composed of small cells located in the region of the superior olivary complex, near the dorsal edge of the accessory olivary nucleus and the trapezoid body, and they are directed dorsally toward the

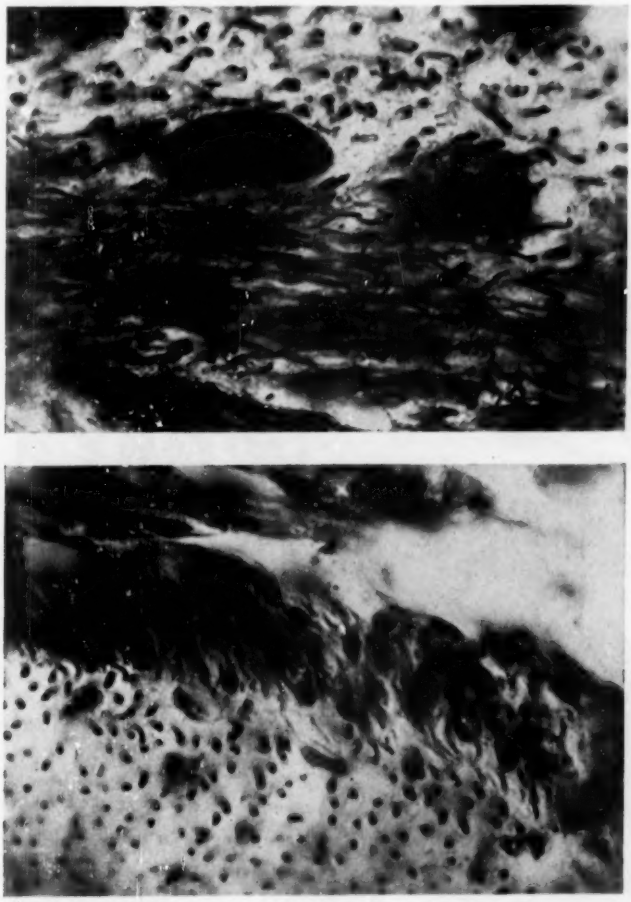

Fig. 2.—*A*, "efferent" fibers (lower part of plate) in Scarpa's ganglion (of which two nuclei can be seen); fine and dense, contrasting with vestibular fibers (upper part of plate), thick and far apart; adult guinea pig. *B*, "efferent" fibers along cochlear nerve; sensory fibers are thick and far apart; efferent fibers are of a lighter color, fine, and dense and show numerous Schwannian nuclei; adult guinea pig. Photomicrographs: compound ocular objective, oil immersion, apochromatic; Leitz.

deep facial knee of the same side, all of them crossing the median line and emerging from the medulla between the vestibular nerve and the nervus intermedius, and then continuing between the two portions of the vestibular nerve. Rasmussen

stated that this bundle continues its course in the cochlea by way of the spiral bundle of the Corti ganglion, a statement that the technique of Weber has made it possible for us to verify.

RADICULAR PORTION

On level with the radicular portion of the seventh pair of cranial nerves, the fibers are grouped in a large bundle. Certain preparations reveal the apparent ori-

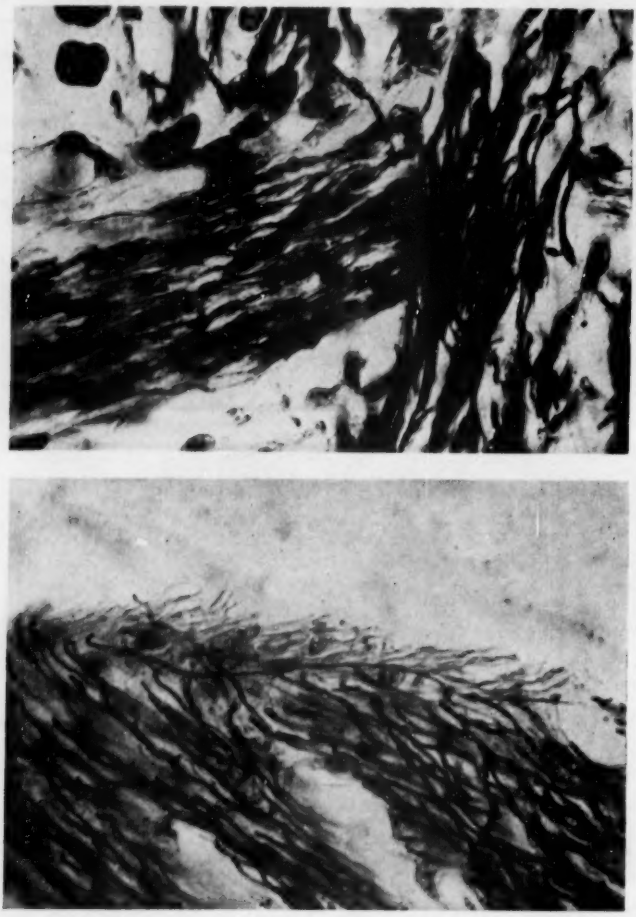

Fig. 3.—*A*, spiral bundle (light-colored fibers, yellow, fine, dense, numerous Schwannian nuclei), perpendicular to auditory dendrites, which are black, thick, and far apart. Two nuclei of Corti's ganglion can be seen in the left upper part of the plate. Young cat. *B*, third spiral bundle near foramina nervosa: efferent fibers, light-colored, fine, dense, and forming a bed, perpendicular to the sensory fibers, which are black, thick, and far-apart. Young cat. Photomicrographs: compound ocular; objective, oil immersion, apochromatic; Leitz.

gin of this bundle at the point where it emerges from the surface of the medulla and crosses the pia mater, between the vestibular vein and the nervus intermedius.

Very soon this dense mass of thin nerve fibers passes between the superior and the inferior portion of the vestibular nerve. It then courses behind the inferior portion of the latter and continues along the posterior wall of the saccular portion of Scarpa's ganglion, which separates it from the cochlear nerve above. Some of these fibers may gather in little bundles, which pass into the mass of the vestibular ganglion. On level with this ganglion the bundle becomes smaller, with absolutely no relay; finally, it turns around the external extremity of the saccular portion of the ganglion in order to take an oblique course in front, outside, and above, thus cros-

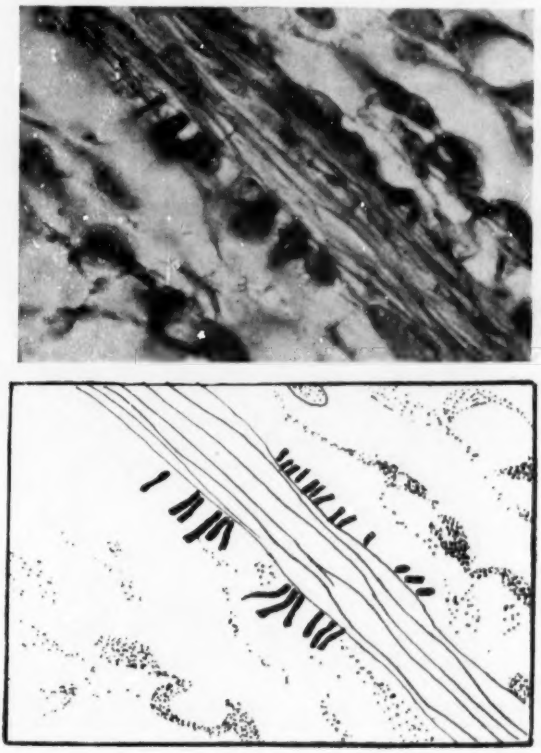

Fig. 4.—Embryo of guinea pig, 26 mm. Section of cochlea between ganglion and organ of Corti, perpendicular to sensory dendrites (black and thick) and tangent to efferent spiral bundle (yellow fibers, light-colored and fine), which appears in longitudinal section. Photomicrograph: compound ocular; objective, oil immersion, apochromatic; Leitz.

sing the inferoexternal face of the cochlear nerve, to which it adheres, without becoming integrated with it. This situation leads us to believe that this bundle corresponds to the vestibulocochlear anastomosis described by Oort in 1918.[14]

This nerve bundle is never increased in size at any point by a contingent of fibers from the nervus intermedius. The fibers being at first myelinated, their histological aspect changes where the bundle rejoins the cochlear nerve and penetrates the modiolus. These fibers give off many very fine collaterals, which accompany

them afterward in a parallel course; the nuclei of Schwann cells increase in number, the fibers then varying in size at one point or another along their course and their myelinated sheaths disappearing.

COCHLEAR PORTION

At the point where the bundle, adhering to the lower face of the cochlear nerve, leaves the saccular portion of Scarpa's ganglion, it passes with the cochlear vessels and separates into three or four small branches running toward the first turn of the spiral base, up to the level of the round window. These fibers cross the spiral ganglion (Corti's ganglion); they slip between the cells and remain as a group,

Fig. 5.—Section perpendicular to axis of columella in a young cat. From bottom to top: Corti's ganglion; spiral efferent bundle; sensory dendrites perpendicular to efferent fibers; (a second and third spiral bundle can be seen on the same level with these fibers); organ of Corti. Photomicrograph: ocular compound; objective, low apochromatic; Leitz.

accompanied by their lemmoblastic cells. There are several small nerve ramifications in the median part of the spiral canal of the modiolus, but the fibers converge in crossing Corti's ganglion to reunite on the external edge of the canal; then, at this point, taking an anterior direction, perpendicular to their first orientation, these fibers form the beginning of the spiral bundles of Corti's ganglion. Situated outside the ganglion with respect to the axis of the modiolus, these bundles adopt a spiral course, perpendicular to the radial direction of the sensory fibers; they wind in this position along the first turn of the spiral.

The bulk of this efferent nerve winds around the lower external face of the cochlear nerve and divides into three branches, which, by a radial direction, joins

with Corti's ganglion. In the cat, for example, the first nerve branch joins the ganglion at the junction of the first and the second quadrant of the first turn; the second, in the middle of the second quadrant (anterior quadrant); the third, about the middle of the third quadrant (superior quadrant of the same turn). Then they cross the ganglion of Corti without relay and, by taking a direction perpendicular to their former one, come to enlarge the spiral bundle, whose origin, at the expense

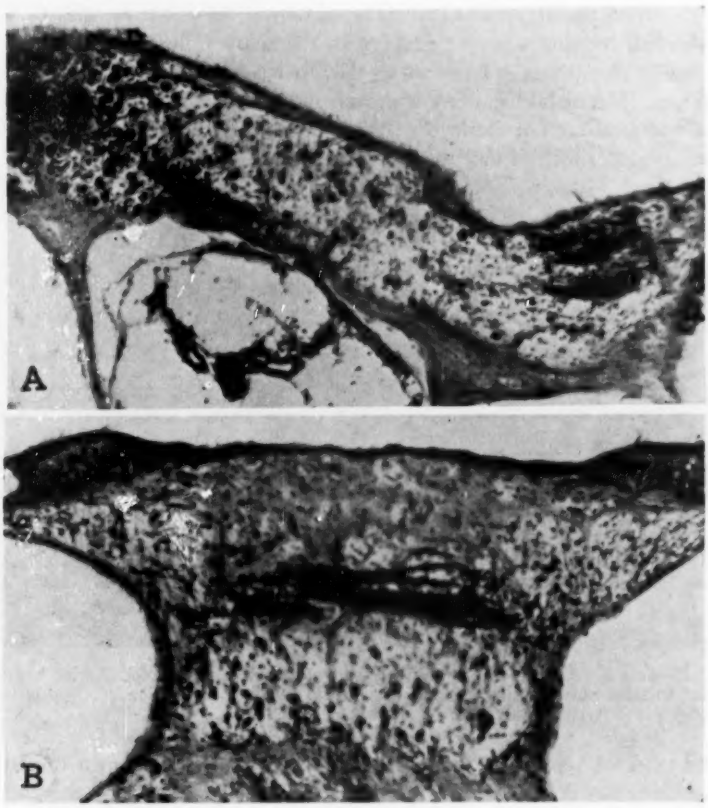

Fig. 6.—*A*, spiral canal of modiolus of cochlea of a guinea pig subjected to noise (137 phons, dominant frequency at 3000 cps) for 20 hours. Right: Sensory zone destroyed; dendrites and nerve cells of spiral ganglion no longer exist. Left: Less affected sensory zone; efferent spiral bundles are undamaged, even in the zone where afferent elements have completely disappeared. *B*, spiral canal of modiolus of cochlea of a guinea pig subjected for 400 hours to same noise as that shown in *A*. All afferent elements are destroyed; only spiral fibers, cut lengthwise, are undamaged.

of the first efferent branches, has been seen. Before the end of the first turn of the spiral, therefore, all the fine fibers are situated outside the ganglion, coiling in the direction of the helicotrema.

From this basal turn, the bundle splits up, for it has on its convexity (external aspect) some fibers, isolated or in small groups, which, tangling for a short moment

with the sensory radial dendrites and then again taking their spiral direction, go on to form a second spiral bundle, parallel to the first, and more external than the first in the cat and above or below it in the guinea pig and the rat.

In the same manner, a third bundle forms, and then a fourth. The last two are composed of thinner fibers, which, having given off some collaterals nearer their end, are reduced in number and lie perpendicular to and under the sensory dendrites in the osseous spiral lamina, very near the foramina nervosa and the internal spiral plexus of the organ of Corti. The section of these last bundles is not oval but extended, creating a carpet underneath the sensory fibers.

A similar disposition is found along the other coils of the spiral right up to the helicotrema. Meanwhile, as they approach the apical region, the bundles become smaller and smaller. On the level of the apex, the last efferent fibers mingle with the sensory dendrites and take the same direction as they do.

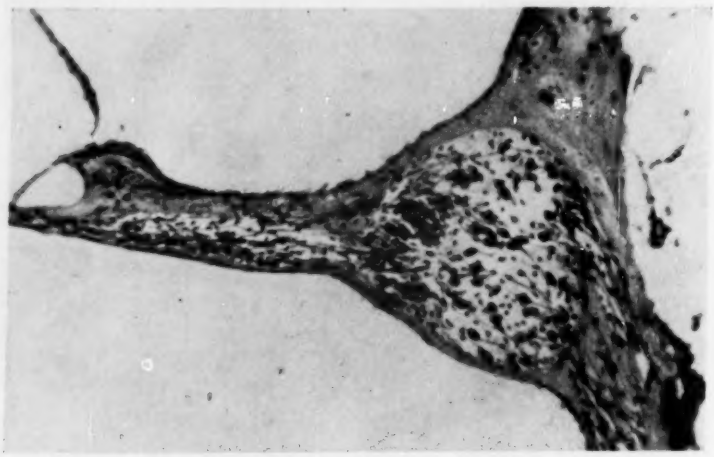

Fig. 7.—Cross section of organ of Corti and of spiral canal of modiolus of a guinea pig subjected to same noise as that shown in Figure 6, showing destruction of sensorial elements. The bundles of efferent spiral fibers remain undamaged.

The external spiral bundles of Corti's ganglion are well known to anatomists and histologists. Boettcher [4] and Kolliker had described them by the end of the last century; then Ramón y Cajal,[5] Retzius,[23] and Held [9] gave them as essentially sensory character, admitting the presence of certain dendrites of the cells of the Corti ganglion with a radial course at first and ending in the organ of Corti, which was not on the same radius as the corresponding cells of the ganglion. The numerous fibers following this course, at the various levels, thus constitute, by virtue of their spiral segment, the spiral bundles of Corti's ganglion.

Later, Lorente de Nó † observed that, although the majority of the fibers of this bundle are sensory fibers, there exist among them some special fibers of unknown origin and destination that do not proceed from the cells of the ganglion.

Weber's technique has shown that there are rare sensory dendrites mingling with the spiral bundles, as illustrated by the schema of Ramón y Cajal, and that

† References 12 and 13.

the great majority (more than 98%) of the fibers of this bundle are composed of the special fibers that one can follow from their bulbar origin and that have nothing in common with the spiral ganglion of Corti, at the level of which we have never seen a single fiber of an "efferent" contingent making a relay.

The efferent nature of these fibers was confirmed by the fact that acoustic trauma does not affect them as it does the afferent fibers. Even after 500 and 1,000 hours of exposure to loud tones of 137 phons, the spiral bundles of guinea pigs

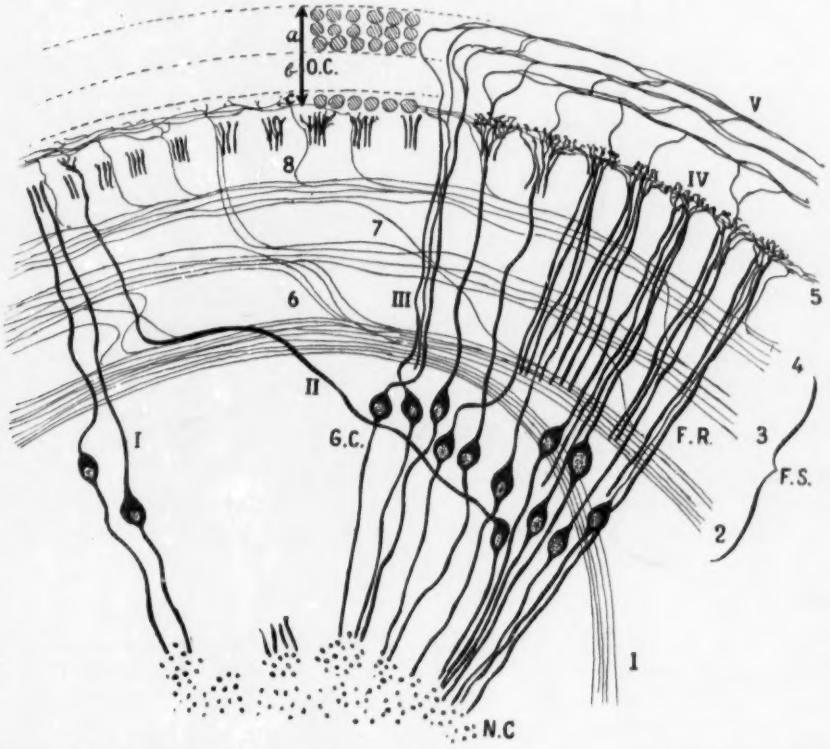

Fig. 8.—Spiral fibers on the level of the third quadrant (upper quadrant) of the first cochlear spiral of a young cat (half schematic): *O.C.*, organ of Corti (*a*) external cells, (*b*) tunnel of Corti, (*c*) internal cells; *G.C.*, spiral Corti's ganglion; *F.R.*, radial bundles; *N.C.*, section of the central part of the cochlear nerve on level with modiolus.

Fibers, fine and light-colored ("efferent" fibers): *1*, portion of efferent nerve rejoining the first turn on this level; *2*, first spiral bundle; *3*, second spiral bundle; *4*, third spiral bundle near foramina nervosa; *5*, efferent spiral bundles situated in the internal spiral plexus; *6*, "efferent" fibers running from first to second spiral bundle; *7*, "efferent" fibers running from second to third spiral bundle; *8*, "efferent" fibers running from third spiral bundle to internal spiral plexus of the organ of Corti, where they end.

Fibers, thick and black (sensory fibers): *I*, radial fibers; *II*, fibers becoming spiral during their course; *III*, fibers running to the external spiral plexuses; *IV*, internal spiral plexus: *V* external spiral plexuses.

show a nearly normal morphology, although there does not subsist a single sensory cell, a single afferent nerve fiber, or a single nerve cell in the spiral ganglion.[19] These conclusions correspond to those recently published by Fernandez [8] and are

evidence that, in spite of the intensity and duration of the stimulus, these fibers do not function beyond their potentialities, as is the case in sensory innervation.

On the other hand, these observations do not agree with the recent conclusions of del Bo and Livan (1951),‡ who have described, at exactly the same level as the second, third, and fourth spiral bundles which we observed on our sections, prepared by the Weber technique, the plexus which they named "paramarginal plexus" and "marginal," and which they considered to depend for the greater part on the sensory dendrites.

TERMINATION OF FIBERS

At each level of the various turns of the spiral, fibers, or collaterals, detach themselves from the spiral bundles, mingle with the sensory ıbers, and end with them in the organ of Corti on a level with the internal spiral plexus, where they resume an apical spiral course of a certain length and terminate in the inner hair cells. This spiral segment of their course corresponds in part to the bundle located under the inner hair cells of the organ of Corti, only dimly recognized by Retzius, then better described by Held [9] and Poliak,§ and, finally, established by Lorente de Nó,‖ but the interpretation of these authors was that this bundle was mostly formed of sensory fibers.

None of these fibers seem to reach the region of the stria vascularis.

One can therefore affirm that the inner hair cells have a double innervation: an afferent innervation, formed of radial fibers, and an efferent innervation, composed of spiral fibers.

As for the outer hair cells, they are almost exclusively innervated by the well-known afferent fibers, radial and spiral. A description of these terminations, recently published (1951),[20] was also the subject of a very thorough research by Fernandez [8] which was reported at the same time. This author, after studying the cochlear canal of a guinea pig previously dissected, reached similar conclusions.

COMMENT

In short, we can affirm that the organ of Corti is innervated by a double system of nerve fibers: the sensory fibers, already known, and the efferent fibers. The latter constitute a bundle clearly described in its bulbar portion as early as 1942 (Rasmussen [21]). Weber's technique of fixation and silver impregnation has made it possible for us to determine with precision the peripheral topography of the fibers and their terminations at the level of the inner hair cells of the organ of Corti (Portmann and Portmann [20]). These conclusions were confirmed by Fernandez [8] in a similar research.

It it thus possible to bring together in one general schema histological observations so far isolated and, for the most part, morphologically correct, such as the vestibulocochlear anastomosis of Oort, the spiral bundles of Boettcher, and the spiral fibers of the internal plexus of the organ of Corti, described by Retzius, Held, Poliak, and Lorente de Nó, whose common membership in the same efferent system had never been demonstrated before.

‡ References 6 and 7.

§ References 15 and 16.

‖ References 12 and 13.

These fibers, therefore, constitute in the cochlea bundles that are anatomically well individualized; they come from the medulla, make their way between the two portions of the vestibular nerve, and, after having rejoined the cochlear nerve, coil in a spiral around the ganglion of Corti up to the helicotrema, terminating at the various turns, leveling with the internal spiral plexus.

The possibility of following their course on perfect serial sections from the medulla to their ultimate terminations permits the observation that they do not present any relay along their course, particularly in the Scarpa and Corti ganglions. Since this observation does not conform to the usual schema of the vegetative system of Langley and Gaskell, it is difficult to accept completely the nature of these fibers as parasympathetic.

Their efferent nature is, nevertheless, certain, as their topography, their morphology, and, finally, their behavior under acoustic traumatism attest.

1. *Topography.*—The fibers have no cell bodies along their course, particularly as they pass through the ganglions of Scarpa and Corti, a disposition which is not characteristic of sensory fibers.

2. *Morphology.*—These fibers are finer than the sensory fibers, are packed tightly together, without a myelinated sheath over a large part of their course, and, finally, present many Schwann nuclei, these characteristics being specific features of the efferent fibers of other nerves. Lastly, they appear lighter in color than the black sensory fibers and are of a brown, or even yellow, coloration.

These tinctorial facts present additional proof in favor of the difference in function of these two systems, as is shown by the recent research of Baud and Pernoux,[1] carried out with the use of the electronic microscope.

It is known that the ultrastructure of the nerve fiber includes protofibrils and interprotofibrillar spaces, the proportions varying according to the function of the nerve. Baud and Pernoux[1] observed that, thanks to the technique of Weber, the silver granules penetrate within the nerve fibers and deposit themselves between the protofibrils; thus, the density of the fibers, that is, the intensity and quality of their coloration, depends directly on the ultrastructure of the element, and thus on its function. For instance, the sensory fibers, with large interprotofibrillar spacing, appear very dark, and the organovegetative fibers, with reduced spacing, appear light-colored.

On our sections, the spiral fibers were always of a lighter color than the sensory fibers, in whatever part they were observed and, above all, in whatever species (rat, cat, or guinea pig) they were studied, proving that their ultrastructure is different and, consequently, that they do not play the same part.

3. *Behavior Under Acoustic Trauma.*—The spiral fibers remained unchanged b[illegible]ensory fibers degenerated when subjected to acoustic trauma.

CONCLUSIONS

All these facts are formal proof of the efferent nature of the fibers and point the way for future physiological research concerning them. The function of these fibers has yet to be established; however, we believe with Fernandez that they constitute the anatomical substratum of cochlear interactions.

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SOME LARYNGOTRACHEAL COMPLICATIONS ASSOCIATED WITH ENDOTRACHEAL ANESTHESIA

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RECENT years have seen a great increase in the use of endotracheal intubation, which has enhanced both the safety and the smoothness of anesthesia. Despite the undoubted advantages of this technique, the dangers, however rare, which accompany it should not be underestimated. These, for the most part, are connected with the reaction of the mucous membrane of the upper respiratory tract.

Since 1932, when Clausen [1] described the first case of laryngeal granuloma following intubation, the number of cases reported has risen steadily, but relatively little has been written on other complications. It is probable that this scarcely reflects an accurate picture of the complications which do, in fact, occur.

Flagg's survey,[2] in which he received responses from 154 laryngologists, 99 of whom were called for consultation because of injury to larynx or trachea following endotracheal intubation for anesthesia, supports this belief; 34 of these reported a total of 101 granulomas. Other special injuries mentioned were paralysis, hemorrhage, and lacerations of the vocal cords; laryngotracheal edema; leucocytic membrane of cord and trachea; tracheal webs, and perforation of the trachea.

In this connection we present the following five cases in which symptoms were so severe that relief could be obtained only after bronchoscopy or tracheotomy.

REPORT OF CASES

CASE 1.—F. C., a woman of 45, had a nephrectomy performed on Dec. 21, 1952. Premedication was morphine sulfate, 0.01 gm., and atropine, 0.0005 gm., given an hour before operation, which lasted for two and a quarter hours. Induction was with pentothal, and the throat was sprayed with 2% tetracaine (Pontocaine) hydrochloride and cocaine before intubation, which was fairly difficult. The patient was placed on her left side and anesthesia maintained with nitrous oxide-oxygen-ether. Twelve milligrams of tubocurarine was given during the operation.

The next day there was marked difficulty in breathing, accompanied by stridor. Indirect laryngoscopy showed a normal epiglottis; the area around both arytenoids was only slightly swollen, and ulceration had occurred on the right with the formation of a whitish fibrinous deposit. There was marginal erosion of both vocal cords, while in the subglottic region a whitish gelatinous membrane almost obstructed the lumen, leaving only a tiny gap. Tracheotomy was performed, with immediate relief of symptoms, but the patient's eventual recovery was further complicated by a small pulmonary infarct on the right side.

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CASE 2.—C. H., a girl of 2½ years, suffering from pes cavus, underwent a corrective operation on Jan. 7, 1953. Premedication consisted of atropine, 0.5 mg., and meperidine, 25 mg., given an hour and a half before operation. Induction was by ethyl chloride and open ether, and intubation was easily performed. The child was placed in the prone position. Anesthesia was maintained by nitrous oxide-oxygen-ether and trichloroethylene (Trilene) on a semiopen circuit for two hours.

After operation the child developed a slight inspiratory stridor, although her voice remained clear. Steam inhalations and antibiotics were ordered, but the child's condition deteriorated. On the following day there was severe dyspnea, with cyanosis, inspiratory stridor, and wheezing; there was no hoarseness. Direct laryngoscopy revealed nothing abnormal in the epiglottis and vocal cords, but there was a gray glutinous membrane in the subglottic region. The child's condition deteriorated the next morning, and she became hoarse. Direct laryngoscopy showed that the membrane had spread across the subglottic region and had become adherent to the under surface of the cords, leaving only a very small opening. Tracheotomy was therefore performed, and the child's respiration and color immediately improved. A culture was taken from the membrane but proved sterile. Culture of a carious tooth grew Staphylococcus albus. On Jan. 14, there were coarse râles in both lungs. X-ray showed atelectasis of the whole left lung, which rapidly improved. On Jan. 22, direct laryngoscopy showed a normal larynx and subglottic region, and the cannula was removed.

CASE 3.—E. C., a woman of 30 years, underwent a conservative myomectomy on Feb. 11, 1953. She was given morphine sulfate, 0.01 gm., and atropine, 0.0005 gm., one and a half hours before operation. Induction was with pentothal. The throat was sprayed with 2% tetracaine and cocaine and a highly traumatic intubation performed. Maintenance was with nitrous oxide-oxygen-ether. Immediately after the operation it was reported that the patient had vomited blood.

The next day the patient had a temperature of 38 C. (100.4 F.). There were no respiratory symptoms. The following day (Feb. 13) there was respiratory distress, and râles were found at the left base. An x-ray of the chest was negative. On Feb. 14 respiratory distress increased. Indirect laryngoscopy on Feb. 15 revealed a normal epiglottis; there were fibrinous threads between the vocal cords, restricting their movement and holding them in adduction. The condition rapidly deteriorated, and tracheotomy was performed. Material was sent for culture, which later revealed a scanty growth of Staphylococcus aureus resistant to penicillin, streptomycin, and chloramphenicol (Chloromycetin). Respiratory distress was immediately relieved, but on Feb. 22 some membrane was still to be seen in the subglottic area. Decannulation was finally performed on March 3, 1953.

CASE 4.—R. J., a girl aged 5 years, had a ligation of a patent ductus arteriosus performed on May 5, 1953. Premedication consisted of secobarbital sodium (Seconal), 0.03 gm., given two hours before operation and morphine sulfate, 0.003 gm., and atropine, 0.0005 gm., given one hour preoperatively. Induction was with pentothal. Intubation was easily performed after topical anesthesia with 2% tetracaine and cocaine. The child was placed on her right side and anesthesia maintained for four hours with nitrous oxide-oxygen and traces of ether. Six milligrams of curare and 25 mg. of meperidine were given during anesthesia. Two and a quarter hours after induction of anesthesia, there was copious reflux vomiting of undigested food, which was removed by suction. At no time during operation was the endotracheal tube obstructed by vomitus. At extubation there was a moderate amount of mucus, and a careful tracheobronchial toilet was done. Some hours after operation there was again copious vomiting of undigested food and black fluid (resembling coffee grounds). The child was cyanotic, and respiration was labored at a rate of 40 per minute in spite of the oxygen tent. The condition was further complicated by subcutaneous and mediastinal emphysema. Direct laryngoscopy revealed a normal epiglottis except for very slight redness. The vocal cords were normal. In the subglottic space some swelling and fibrinous deposit were seen, which slightly obstructed the lumen; this was cleared of fibrin by suction, and respiration was somewhat relieved. An x-ray of the chest revealed atelectasis of the right upper lobe. The next morning, because of recurrent respiratory distress, direct laryngoscopy was again performed and revealed a gray-white membrane below the vocal cords. Bronchoscopic examination showed that in the right main bronchus there was a small membrane, which was removed. Respiration was temporarily improved after this, but tracheotomy had eventually to be performed. A culture of material obtained at this time revealed

Staph. aureus. Steam inhalations, oxygen, and antibiotics were continued, and the child's condition gradually improved. The cannula was removed on May 28, 1953, and the atelectasis eventually resolved.

CASE 5.—M. G., a boy aged 5 years, had a corrective operation for strabismus on June 11, 1953. Preoperative medication consisted of morphine sulfate, 0.004 gm., and atropine, 0.0005 gm., given one and a half hours before operation. Induction of anesthesia was with vinyl ether (Vinethene) and open ether. An easy intubation was performed, but some difficulty was experienced in extracting the stylet. Anesthesia was maintained with nitrous oxide-oxygen and ether on a semiopen circuit for one hour and fifteen minutes. A few hours after operation the child became restless, and stridor with suprasternal retraction on inspiration was noted. There was no hoarseness or tachypnea. The pulse rate was 100 per minute. Cyanosis was present when the child was excited. Direct laryngoscopy revealed hyperemia of the laryngeal mucosa; the vocal cords moved freely and were covered with a small amount of whitish exudate. There was no marked edema of the epiglottis or other structures. Steam inhalations, antibiotics, and oxygen, when necessary, were ordered. The next day respiration was much improved, but stridor and suprasternal retraction were still present. Conservative treatment was continued till June 14, 1953, when direct laryngoscopy was again performed. This revealed a white subglottic membrane, which was dislodged by the bronchoscope and caught by the metal suction tube. This was followed by slight improvement in respiration. A second bronchoscopy with suction had to be performed on June 18, 1953, because of respiratory distress and recurrence of the subglottic membrane. Microscopic examination of the material removed revealed a membranous structure comprised of fibrin and red blood cells. A culture revealed a nonhemolytic Streptococcus and a Staph. aureus sensitive to chloramphenicol only. Although there was initially a marked improvement following bronchoscopy, this was not maintained, and tracheotomy was eventually performed on June 19, 1953. Recovery was uneventful.

COMMENT

It does not appear farfetched to assume the possibility of trauma when a tube is introduced into the larynx and trachea and left to lie as a foreign body against the delicate mucous membrane. In a study of a large number of patients after endotracheal anesthesia, evidence of trauma was found in a high percentage by Donnelly,[3] Wylie,[4] and Baron and Kohlmoos.[5]

Different authors have incriminated varying factors as a cause of injury. Among these are the mechanical trauma of introducing a tube, the use of heavy or unsuitable laryngoscopes, and the use of a tube of too wide a bore [2] or of a shape which is anatomically incorrect.[6] Other factors held responsible are faulty positioning and hyperextension of the head [7] and the length of time the tube is allowed to stay in place. Allergic reactions to lubricants have been described.[8] Some authors hold that a tube which is too mobile may be a source of damage.* The possibility of infection is also seriously considered, particularly in association with deficiency states, debility, and upper respiratory infection. The sensitive nature of the mucosa in children has been stressed by others who believe that the loose areolar tissue is particularly susceptible to injury reaction, which is all the more striking in view of the normally narrow subglottic funnel.†

It would thus appear from these many factors that endotracheal anesthesia is not without its dangers. In two of our own cases, mechanical trauma at the time of introducing the tube was a marked factor. With regard to the lubricant, we have used 1% cyclomethycaine (Surfacaine) ointment in all our endotracheal cases, and in none have we found signs of generalized sensitivity, although we are unable

* References 6 and 7.

† References 9 to 11.

to express an opinion on the probability of a purely localized reaction. In four of our cases the tube was in place for less than two and a half hours and in one for about four hours. When one considers that many of our patients retain tubes for six to eight hours without subsequent complications, we are not inclined to stress the time factor as a cause of trauma.

Two patients (Cases 2 and 4) presented potentially infective elements—the first having a very carious tooth and the second displaying reflux vomiting during and after operation.

In all five cases the picture was characteristic and similar to that described by Etsten [12] and Baron and Kohlmoos.[5] There was no marked edema, but a grayish-white fibrinous membrane covered the subgottic area. With so few cases at our disposal we are unable to explain why these particular cases were involved, but it is our impression that any single factor should not be held responsible. It might well be argued that trauma and infection together are probably primary causes but that the other factors play an additional part.

It will be noted that all of our cases occurred in women and children, a finding which is in keeping with that of other authors.‡ The larynx in these cases is relatively small, and we believe that there is a tendency to attempt to use a tube of too wide a bore, with subsequent damage to the laryngeal area, which, as has already been stated, is particularly sensitive in children. It is worth considering whether this may not be the case with women as well. Insufficiently lubricated tubes may also be a source of trauma. The traumatic element can be minimized by improved technique, such as attention to positioning, full relaxation of the oropharynx and larynx, and the gentle use of light suitable laryngoscopes and of tubes of correct size. In this connection we believe that the use of a stylet, while extremely valuable, presents a danger of its own when roughly introduced or extracted (Case 5). Contamination may be avoided by careful preoperative examination and toilet of the nose, mouth, and pharynx and scrupulous attention to the cleanliness of laryngoscopes, tubes, and suction catheters. Postoperatively the patient should be carefully watched for hoarseness and stridor, and early treatment by steam inhalations, oxygen, and antibiotics may be of value. In our five cases tracheotomy was performed, although in two (Cases 4 and 5) temporary relief had been obtained by bronchoscopic suction. Our decision to perform tracheotomy was governed by the undesirability of adding to the physical and psychic stress contigent on recurrent obstruction and subsequent manipulation of an already traumatized larynx and trachea.

During the past two and a half years over 2,500 endotracheal anesthesias were administered in our hospital. In addition to the five cases reported, there was one case of severe dyspnea after endotracheal anesthesia in a child, which was relieved by conservative treatment. In two adult patients, both women, laryngeal granulomata developed a few weeks after endotracheal anesthesia.

Although the number of serious complications is small, they should by no means be underestimated. We do not believe that one should refrain from the use of endotracheal anesthesia, but we feel that more attention should be paid to precautions and that the potential causes of laryngotracheal irritation and injury be

‡ References 5 and 12.

eliminated as far as is feasible. Endotracheal anesthesia should not be used indiscriminately, and special attention should be given to the indications for this technique in children.

SUMMARY

Five cases of subglottic membrane following endotracheal anesthesia are reported. Various potential causes of this complication are reviewed and discussed. Attention is particularly drawn to the occurrence of this complication in children and women. The suggestion is made that recurrence of respiratory obstruction following bronchoscopic treatment calls for early tracheotomy. The opinion is expressed that endotracheal anesthesia should not be used indiscriminately, and more precautions are recommended in order to avoid laryngotracheal complications.

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TREATMENT OF NODULES OF VOCAL CORDS BY CHEWING METHOD

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NODULES of the vocal cords is an affliction that is as disquieting to the patient as it is puzzling to the laryngologist. Ever since Tuerck first described the condition, in 1868, discussion of the etiology, the histological nature, and the therapy has not ceased. Generations of laryngologists have argued whether the nodules are genuine tumors, whether they are mechanical irritations, or whether, as Fraenkel [1] suggested, they originate in the surface glands. As late as 1933 Nadoleczny [2] stated that "the development of singer's nodes is even today not completely understood." The voluminous literature can be found in the bibliography of Tarneaud's [3] monograph.

General agreement has been reached on the assumption that nodules of the cords are invariably the result of misuse or abuse of the voice. This very fact places the nodules in a special category. They are visible organic changes that are the consequence of a functional disorder.

The term "singer's nodes" is not very appropriate, since they occur in all groups of the population. They are frequently seen in children who abuse their voices and in adults who use the voice professionally—public speakers of all kinds, teachers, actors, and, of course, singers. In the latter group nodules are more frequently seen in those with high voices—tenors, sopranos, and particularly coloraturas. Very often the development of nodules is the penalty a singer pays for singing habitually above his natural range. The baritone who forces his voice into the tenor range and the mezzo-soprano who sings soprano parts belong to this category. As a rule, the condition is seen more frequently in women. As Tarneaud * suggested, the greater frequency of vibration of the cords in higher ranges multiplies the harmful effects of vocal misuse.

The diagnosis of nodules is, of course, easy once the patient is seen by a laryngologist. The patient comes to the laryngologist or logopedist because of some impairment of vocal function, usually of long standing. The voice is frequently hoarse, breathy (with waste of air), and easily fatigued. The singer will complain about uncertainty of soft notes, particularly in the middle range.

While the functional character of the nodules has been generally accepted, no such agreement has been reached about the therapy. Three approaches have been stressed in varying combinations—voice rest, surgery, and vocal reeducation.

Unfortunately, the accent in the recommendation of most authors is on voice rest. A review of the textbooks of laryngology and logopedics reveals that most

* Tarneaud,[3] p. 91.

authors stress the need for complete voice rest. To mention a few, St. Clair Thomson [4] feels that "strict silence should, if possible, be insisted on, and it may require months of absolute dumbness in some cases before the nodules will completely disappear." Ballenger [5] recommends "refraining from singing and speaking." Lederer and Hollender [6] advocate "rest as the first requisite." The latter three authors add that speaking and singing should be resumed only after correction of faulty methods of voice production.

Of the authors of logopedic textbooks, Luchsinger [7] feels that "in all cases prolonged voice rest is necessary, extending to speaking, clearing of the throat, and whispering." West [8] declares that "the best treatment is a complete rest for the voice, no singing, no talking, not even laughing."

Now, voice rest, as such, does not solve anything. In children, it is almost impossible to enforce. To the professional speaker, the command of prolonged silence means a catastrophe that deprives him of his livelihood and threatens to transform him into a neurotic who trembles for his voice. Even in the singer, an intensive retraining of the voice, which begins right after the diagnosis has been made, is definitely preferable to a period of complete silence that has no value as such and might be outright dangerous. Tarneaud † is right in saying that "in functional troubles the laryngologist will not obtain a good result by simple voice rest if it is not followed by reeducation, which will be more difficult the longer the rest is prolonged, just as with the athlete the necessary retraining is harder after a long rest." To expose the vocal organs to the danger of muscular atrophy by inactivity goes against all principles of modern functional rehabilitation.

The next approach to be considered is surgery. Wolf [9] advocates surgery if voice rest fails; so do Ballenger,‡ Jackson,[10] Tarneaud,§ and Moses,[11] who feel that removal of the nodes, followed by vocal reeducation, is preferable to the doubtful results of voice rest. Now, it cannot be denied that in the case of large nodules of long standing surgical removal is necessary before vocal training is undertaken. But in the case of small nodules, which are more frequently seen, the surgical treatment is doubtful and risky. Even in the best hands it is a delicate procedure, and the psychological trauma of such an operation is considerable. The speaker or singer who undergoes it will focus the blame for failure in his chosen career on the operation instead of on his poor vocal equipment and artistic shortcomings, where the blame belongs.

In any case, the treatment of nodules should be centered around removal of the underlying cause, the functional disorder. At this decisive point the recommendations tend to become rather vague. The laryngologists, who, as a rule, are on unfamiliar ground if faced with functional disorders, usually limit themselves to the general advice of vocal reeducation. They leave it to the teacher of singing or dramatics to find the solution and are satisfied to control the progress, if any is made, by occasional examinations.

A solution can be found only in the teamwork of the laryngologist and the voice therapist who is able to analyze the mistakes of vocal function that have led to the development of nodules. West ‖ has stated the problem correctly in stressing the

† Tarneaud,[3] p. 101.
‡ Ballenger,[5] p. 428.
§ Tarneaud,[3] p. 105.
‖ West,[8] p. 141.

search for a level of optimum resonance and for a natural pitch "at which the greatest amount of the energy carried in the air column is transmitted into sound waves."

It is our opinion that the chewing method, as described by one of us (Froeschels),¶ is an excellent tool in dealing with functional disorders that have led to the formation of nodules. The chewing method makes use of the far-reaching identity of chewing and vocal functions. Limitation of space forbids description of the steps of the chewing therapy. As with most methods of vocal training, the requisite of successful handling is a greater familiarity with all details than that which can be gained from a short outline. The technique and application of the method have been described in the above-mentioned papers by Froeschels and in the book by Weiss and Beebe.[14]

The use of the chewing technique corrects the vocal hyperfunction that is common in vocal disorders. It helps to overcome the waste of air that is frequently found in connection with nodules of the cords. And it is instrumental in solving one of the most difficult problems of vocal reeducation: to find the natural level of pitch. As long as this is attempted somehow at random, by the ear alone, and according to the judgment of the therapist, mistakes are bound to happen. The chewed voice has a surprising ability to find the proper level of pitch unaided by outside interference.

REPORT OF CASES

We present six cases of nodules which were treated exclusively by the chewing method, without surgery and without a period of complete silence. The results were obtained by independent treatments of three patients by each of us. The cases are identified by the initials of the therapist. The patients included two children, a housewife, a priest, and two singers.

CASE 1 (F. B.).—E. W., a girl of 10 years, was first seen in September, 1952, after return from camp, where she had done a lot of shouting, cheering, and group singing. The voice was hoarse and rough, with considerable hyperfunction. The vocal cords were congested but had a smooth margin. Avoidance of all vocal exertion and abstention from group singing were recommended. The child was not seen again until April, 1953. By that time the voice had deteriorated considerably. The child talked with a "ship captain's voice"—deep, rough, hoarse, with much waste of air.

On examinaiton of the larynx, nodules were found at the typical place, at the juncture of the middle and anterior third; they were very distinct, jutting out from the inner margin like two tiny buttons. The cords were pinkish; the nose, sinuses, and throat were normal. The functional character of the voice disturbance was again explained to the mother, who this time agreed to voice therapy. The child was permitted to speak with conversational voice, was instructed to discontinue all singing, and was forbidden to yell and shout.

Chewing therapy was started. In the beginning the child was reluctant to accept the idea of voiced chewing and had to use crackers to do "real" chewing. At the third treatment free chewing was achieved and utilized for counting and reading with voiced chewing. Office treatments were given twice weekly. The intelligent and ambitious child was very cooperative in maintaining a schedule of frequent periods of one-minute chewing during the day and in avoiding voice strain. After a month the voice became higher and much clearer. When the cords were examined on May 8, the nodules had diminished in size.

Treatment was continued at weekly intervals until June 22, when the girl went to camp. At that date only a slight thickening was seen at the place of the nodules. The child was sent to camp with the strict instruction to avoid excessive use of the voice and any group singing. She agreed to report to the therapist every two weeks, on the "honor system," the frequency of lapses into yelling and did so faithfully.

¶ References 12 and 13.

After return from camp the child was again seen, on Oct. 2. The voice was quite clear and pleasant. The cords showed no trace of thickening any more.

CASE 2 (E. F.).—B. A., a girl of 6 years, was first seen on June 28, 1951. According to the mother's report, the girl was hoarse for several weeks after a cold. A laryngologist saw a small node on the right cord at the typical spot. The speaking voice was of the hypofunctional (paretic) type, which sounds as though much mucus was accumulated on the cord. The peculiar noise seems to be due to weakness of the closing muscles of the glottis. As a rule, the hypofunctional hoarseness is the consequence of long-lasting hyperfunctional (spastic) hoarseness which is due to pressure in the glottis or in the laryngeal ansatzrohr.

The chewing method worked immediately, the voice being corrected into a clear, even beautiful one. The patient came twice weekly for treatments. On Aug. 9 no node was visible. The child was last seen on Sept. 5. In spite of a cold the voice was clear and pleasant.

CASE 3 (E. F.).—B. S., a 34-year-old housewife, when first seen, on June 17, 1953, gave a history of severe hoarseness which had developed gradually since February. She had consulted several laryngologists, one of whom found a small node on the right cord. She was ordered to speak softly. Finally she was sent to us.

The nose and throat showed no abnormality except a very small uvula, (a condition which is almost regularly seen after a tonsillectomy). The vocal cords were slightly red. At the typical spot on the right cord a small node was visible. The voice was very soft and breathy, giving the impression that the patient was afraid to use her voice.

Under the influence of the chewing therapy the voice became louder and clearer. By July 10 the node had disappeared (as confirmed by examination of the referring laryngologist one week later).

CASE 4 (E. F.).—J. F., a 32-year-old priest, was first seen on Feb. 5, 1953, having been referred by a laryngologist, with a history of "difficulties with his voice." After every sermon he said his voice was "tired." He became afraid that he might be forced to give up preaching. The specialist found small nodes at the typical spot on both cords.

The patient's voice in conversation and in reading was frequently very high; it was sometimes low, but then it was distinctly of the hypofunctional (paretic) type. The diagnosis was made of unfinished voice change or prolonged mutation of the voice.

The chewing method was used. The patient, who came from Vermont and could stay for only three days, was advised to continue voiced chewing at home. On April 16 he was seen again by the referring laryngologist, who remarked: "The nodes have disappeared; the color of the cords is normal; I never saw nodes disappear that fast in my life."

The patient still comes for two or three days of treatment at irregular intervals. He has not had any voice troubles for months.

CASE 5 (F. B.).—Miss T. S., a 28-year-old singer, is a night-club entertainer who presents a singing parody act which involves great strain on the voice; in addition, there is the handicap of singing in smoky and often noisy places. She was first seen in 1949 and again in 1950, on both occasions for mild upper respiratory infections. Examination of the cords revealed no pathology, except the slight reddening and congestion of an acute irritation.

The patient returned in February, 1953, with the complaint of frequent spells of hoarseness, which she attributed to laryngitis. Examination of nose, sinuses, and throat was completely negative. The cords were of grayish-pinkish color. At the typical spot on the inner margin of the cords small nodules were visible. The voice was strongly hyperfunctional, with considerable waste of air. In singing, uncertainty of pitch was noted, particularly in the middle range. The patient was unable to produce clear notes with a soft voice.

The therapy suffered from two handicaps. The patient was very reluctant to accept the diagnosis of nodes as a consequence of misuse of the voice. She expected treatment for "laryngitis." Extremely tense and—for a number of personal reasons—emotionally unstable, and at the same time professionally successful for the first time in her life, she found it hard to accept the verdict of functional disturbance. For this reason she showed little cooperation in getting used to the chewing approach, but she finally accepted the method.

In addition, it was impossible for her to interrupt a rather profitable schedule of night-club engagements. Treatments could be given for three weeks only. At the end of the period the

nodes had barely diminished in size, but the voice had greatly improved. In speaking, the waste of air had disappeared, and the pitch had descended from a high level (about e^1) by about two notes. In singing, the improvement was less conspicuous but still noticeable.

CASE 6 (F. B.).—Miss N. A., aged 26, a dramatic soprano, complained of frequent attacks of "laryngitis," occurring almost weekly lately, and of increasing uncertainty of pitch, particularly in the middle range.

When the patient was first seen, on June 1, 1951, the nose revealed a moderate degree of vasomotor rhinitis (previous allergy tests were all negative). The throat was normal.

The cords were difficult to visualize because of strong reflexes. They were grayish, and slightly congested. On the inner margin there were small nodes visible at the anterior part of the middle third, distinct in appearance, and flattening out into the otherwise straight bands of the cords. On phonation, only the cords touched each other.

The speaking voice showed marked hyperfunction and breathiness. In singing, the low register was best; the head register was shrill and wavering with a strong tremolo, and the middle range was sung with considerable waste of air. The patient had just changed from a rather poor teacher to a very competent one, who, after hearing the defects of the voice, insisted on a laryngological examination.

At a conference of the patient, the singing teacher, and the writer it was decided to stop singing lessons for a month while voice therapy was given. The patient was instructed in the chewing approach to voice production. After two weeks of working with the chewing method, exercises for the improvement of economy of breathing were added.

After a month, singing lessons were resumed. On July 16, six weeks after the beginning of voice therapy, the patient was again examined. The cords had become whiter. Where the nodes had developed, hardly visible thickening of the inner margins persisted. According to the singing teacher, the voice had greatly improved in certainty of pitch, quality of the whole range, and economy of breath. The patient left New York to participate in the Opera Studio of the Tanglewood Musical Festivals. She sang highly dramatic parts in the studio performance. She was seen again on Sept. 18. The cords appeared normal. The speaking and singing voice was clear.

CONCLUSIONS

The six cases represent a cross section of the groups which are exposed to the dangers of vocal dysfunction that may lead to the development of nodules of the vocal cords. The patients were all treated with vocal training by the chewing method. Five of the patients may be considered as cured; the sixth one (Case 5) may be considered improved. The latter case serves as an illustration of the difficulties that surround the handling of these cases, particularly if the patients are artists. Outside conditions, artistic shortcomings (really good singers rarely develop nodules), and psychological factors combine to make the treatment of these patients an undertaking, the success of which cannot be foretold with any degree of certainty.

However, it seems to us that many cases of nodules can be improved or cured without submitting the patients to the ordeal of silence or to the shock and risks of an operation. All six patients in these cases continued to use their voices for normal communication. The professionals abstained from practice and public use of the voice for relatively short periods of time. They were informed—as they should be—of the existence of nodules, but they were not exposed to the crisis that prolonged silence or an operation invariably produces. They were encouraged by a therapy which channeled their energies into the activity of a complete vocal reeducation.

In a functional therapy of the voice it is difficult to analyze the effect of a method with the exactitude of the laboratory or clinical assay of a drug. This much can be said, however:

The chewing approach to voice production normalizes the voice in pitch and in muscular equilibrium. By doing this, it is instrumental in removing the abnormalities

of vocal dysfunction that lead to the development of nodules. By freeing the vocal cords from the constant pressure of hyperfunction, it creates favorable conditions for the disappearance of the nodules, particularly in the early, reversible, stages.

SUMMARY

Six patients with nodules of the vocal cords were treated with vocal reeducation by the chewing method of Froeschels. The nodes disappeared in five cases; the voice was normalized in five cases and improved in one case.

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CHANGES IN THE AUDITORY OSSICLES IN OTOSCLEROSIS

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IT WAS pointed out by Holmgren [1] in 1938 that in certain cases of otosclerosis the mobility of the malleus was either reduced or totally absent. Two parallel hairs inserted in a Siegle-Brüning speculum enabled him to judge the extent to which the handle of the malleus moves under compression and decompression. Holmgren reasoned that ankylosis of the stapes entailed reduced mobility of the incus, which in turn affected the incudomalleolar articulation, resulting in partial or total immobility of the shaft of the malleus.

Subsequently, Nager [3] (1941) and his co-worker, Covell,[2] (1940) established an anatomical foundation for this clinical observation. They examined 46 temporal bones with auditory ossicles from 31 cases of otosclerosis. Covell divided these 46 bones which showed otosclerotic changes into three groups. The first group included 15 bones with complete ankylosis of the stapes. The second group (16 bones) showed only partial ankylosis at the anterior margin of the oval window, while in the third group (15 bones) there was no ankylosis but merely a distortion of the oval-window niche. Covell used as controls the normal ears of four patients in whom the opposite temporal bone showed otosclerotic changes as well as seven other cases in which the otosclerotic foci were in the niche of the round window, in the internal auditory meatus, and in the neighborhood of the facial nerve. Apart from these 11 ears, which composed Group IV, another 21 healthy temporal bones were examined as controls.

In cases of complete ankylosis of the footplate of the stapes (Group I) Covell found, among other things, a posterolateral displacement of the head of the stapes in the direction of the stapedius tendon. Thereby the incudostapedial articulation became abnormally stretched, and the lenticular process was pushed upward. This change in the position of the lenticular process led to an upward displacement of the body of the incus, thereby affecting the incudomalleolar joint. The small articular process ("cog process") of the incus was found to be in contact with the articular surface of the malleus, usually at one point within the joint. The two bones which normally appear to be equally separated by the cartilage were farther apart at the superior and inferior borders of the joint. These changes led to excessive stretching of the capsular ligaments, with concomitant changes in the articular cartilages.

In the second and third groups the displacement of the incus and the articular changes were less severe. When the annular ligament was partially ossified, the footplate of the stapes was pushed in toward the vestibule on its free margin, thereby affecting the incudostapedial articulation.

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Covell observed gross displacement of the ossicles in 53.3% of the cases in Group I and in 25% in Group II, while in Group III no gross displacement occurred. Moderate to slight displacement was observed in 93.4% of the cases in Group III, 75% in Group II, and 40% in Group I. In Group IV no displacement of ossicles was observed. Thus it becomes obvious that there is a close relationship between the extent of otosclerotic changes at the oval-window niche and changes in the articulations. It is these articular changes (particularly those at the incudomalleolar joint) which, according to Nager and Covell, provide the anatomical explanation for the reduced mobility of the malleus, as observed by Holmgren.

Histological examination of the ossicles of the temporal bones showing otosclerotic changes revealed the following in the neighborhood of the malleoincudal joint: increased vascularization, slight fibrosis of the marrow, and structures resembling the so-called "blue mantles." Covell found these changes in 86.6% of the cases in Group I, 56.3% in Group II, 73.3% in Group III, and 54.6% in Group IV, but they were completely absent in healthy temporal bone.

Cartilaginous rests and interglobular spaces in the ossicles are evidence in support of the view that here also preotosclerotic conditions occur similar to those found in the labyrinthine capsule (Meyer,[4] Oesterle [5]). Nevertheless, it is only very rarely that one encounters otosclerotic changes in the ossicles which resemble those observed in the labyrinthine capsule, i. e., circumscribed patches of newly formed spongy bone sharply demarcated from the old compact bone. As far as I have been able to ascertain, only two such cases have been described. Covell found among the above-mentioned 46 ostosclerotic ears only one with typical otosclerotic changes in the head of the malleus and in the incus. The second case was described by Nager [6] in 1944. In this instance the petrous temporal bone came from a myxedematous patient who had been under observation for years. In the right temporal bone small circumscribed otosclerotic foci were found at the lower extremity of the incus and in the head of the stapes. No changes were found in the ossicles of the opposite side or in the capsule of the labyrinth on either side. These patches were situated in functionally mute areas.

Lempert and Wolff [7] were the next to report on changes in the auditory ossicles in otosclerosis. In 1945 they reported on 115 ossicles and in 1949 on an additional 100 ossicles.[8] A study is made of the incus, malleus, and a number of stapedial crura—all removed in vivo in the course of fenestration operations. Of these 215 ossicles, only 9 were normal. In the others pathological changes were observed in the articulations, osseous tissue, marrow spaces, and, particularly, the blood vessels. These changes may be detailed as follows:

Changes in the blood: arterial congestion; venous stasis; clear globules; accumulation of fragmented erythrocytes; sludge; accumulation of polymorphonuclear leucocytes.

Vascular changes: constriction of vessels; tortuosity of vessels caused by (*a*) hypertrophy of endothelial cells and (*b*) varicosities; endarteritis; fibrosis of the marrow spaces.

Bone changes: excessive "blue mantling"; hypervascularity; decalcification; osteofibrosis; osteoporosis; ischemia with empty lacunae; distortion in the shape of the ossicles, owing to irregular bone contours; more or less extensive fibrotic replacement of bone and evidence of altered stresses and strains in the ossicular chain; hyperostosis.

Articular changes: erosion or lipping of cartilage; deposition of calcium; overgrowth of atypical bone; ankylosis of joints.

In two patients who, subsequent to the fenestration operation, had died of other causes, both otosclerosis of the labyrinthine capsule and changes in the ossicles, as described above, were in evidence. The authors were unable, however, to detect typically otosclerotic foci, as they are encountered in the labyrinthine capsule, i. e., foci which show a sharp line of demarcation from the normal bone. Lempert and Wolff believe that it is on account of the nature of the blood supply that diffuse changes are more frequent in the ossicles. While the blood supply of the ossicles is derived from one source or, at the most, two, the labyrinthine capsule is supplied by six or more sources. This explains why in the otic capsule, with its multiple blood supply, a sharply defined area which is supplied by a certain vessel may show pathological changes, while the surrounding bone still retains its normal blood supply and therefore presents a histologically normal picture. This may possibly account for the fact that only two cases have been described in the literature of

Fig. 1.—A specimen with ankylosis at the malleoincudal joint.

ossicles showing the same sharply demarcated otosclerotic foci as occur in the labyrinthine capsule. Lempert and Wolff hold that the pathological changes in the ossicles, as described above, represent an otosclerotic process which does not exhibit the usual characteristic sharply delineated form. Further studies will, no doubt, show whether these vascular and osseous changes are in fact pathological, i. e., whether they are normally absent in healthy ears.

In certain cases of otosclerosis there may occur a deposition of calcium in various places in the auditory ossicles. If calcium is deposited in the interarticular disc, it may lead to ankylosis of the joint in question. Lempert and Wolff [5] reported two cases of ankylosis of the malleoincudal joint—one in a case observed by themselves and one in a specimen supplied to them. They have also described a case of ankylosis of the incudostapedial joint. Since 1949 Lempert and Wolff * have observed 10 additional cases of ankylosis of the malleoincudal joint. In two other cases in which ankylosis was suspected macroscopically, histological examination showed that the restriction of movement was caused by a densely fibrous capsule.*

* Personal communication to the author.

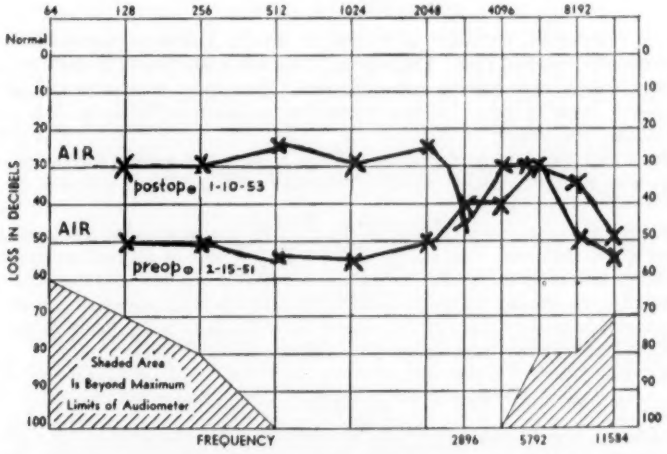

Fig. 2.—Audiogram illustrating preoperative and postoperative hearing of the patient.

Fig. 3.—Photomicrograph demonstrating the histological changes at the malleoincudal joint.

REPORT OF A CASE

Among 35 fenestration operations performed by myself, I have encountered one case of ankylosis of the malleoincudal joint. The patient, L. C., was a 20-year-old woman. Her parents were alive and healthy; their hearing was unimpaired. There were two brothers and six sisters. One of the sisters (aged 18 years) had been operated on by myself in February, 1951, for clinical otosclerosis of the left side.

The present patient had not been hearing well for a year and complained of tinnitus on both sides. There was no vertigo and no paracusis willisiana. A hearing test indicated clinical otosclerosis.

A fenestration operation was performed on the left side. On opening the malleoincudal joint, I found that it was ankylosed. Amputation of the head and neck of the malleus was first performed, and only then could the head of the malleus be removed together with the incus (Fig. 1). Immediately after the opening of the fenestra, the patient's hearing improved, and the gain in hearing has been maintained (Fig. 2). The ossicles were examined by Dr. Wolff at the Lempert Institute of Otology, and ankylosis was confirmed histologically (Fig. 3).

SUMMARY

A review of the literature on the condition of the ossicles in otosclerosis is presented.

A case of ankylosis of the malleoincudal joint is described.

Dr. Wolff performed the histological examination in this case.

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TRACHEOTOMY IN POLIOMYELITIS

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THE IMMEDIATE concern in any case of acute poliomyelitis is the treatment and management of two systems, namely, the cardiovascular and the respiratory. It is through the involvement of these systems that death ensues, even though the actual pathology may be confined to the central nervous system. As stated by Plum and Wolff [1]:

> In our experience, arrhythmias and shock due to failure of neurogenic cardiovascular regulation proved unresponsive to therapeutic measures, such as administration of the digitalis glycosides or phenylephrine (neo-synephrine®) and parenteral injection of fluids. In contrast to the discouraging prognosis offered by circulatory failure, the ill effects of respiratory insufficiency could often be prevented or reversed, and therefore the maintenance of a physiologic respiratory exchange was given primary attention.

Most of the signs, characterizing what we will later refer to as the encephalitic type, could and probably do result from hypoxia (low oxygen tension) and hypercapnia (high carbon dioxide retention) together with whatever local brain damage is due to the virus itself.

Summarily, McDowell and Wolff [2] state:

> The usual indications for tracheotomy in poliomyelitis have been the accumulation of secretions in the tracheobronchial tree, abrupt closure of the airway from laryngospasm, and hypoxia and hypercapnia, with cyanosis, delirium, restlessness, and coma. Through blocking of the airway, hypoxia and hypercapnia may be sudden in onset, dramatic in character, and easily detected by observation. More often the filling of the airway with secretions and symptoms of hypoxia and hypercapnia have a gradual and insidious development, which may often be mistakenly attributed to progression of the infectious process in the nervous system.
>
> The complications that follow development of hypoxia and hypercapnia may seriously endanger the patient's life and may be more damaging than poliomyelitis itself. A prophylactic tracheotomy is one that is done before these complications develop and before the procedure becomes technically difficult because of severe ventilatory failure.

Although measuring the vital capacity, tidal air, and minute volume is a simple procedure and extremely important in assessing respiratory insufficiency resulting from intercostal muscle and diaphragmatic involvement, it is of little value as an aid in determining respiratory insufficiency on the basis of obstruction of the airway. Likewise, oxygen tension determinations are cumbersome and not too reliable. Venous carbon dioxide content determinations may be easily obtained and are a rough gauge as to arterial carbon dioxide tension. Arterial carbon dioxide deter-

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minations are more valuable but are not readily available for clinical use. For these reasons, in setting down a guide for the neophyte as well as the experienced physician to determine when to do a tracheotomy, we must frequently use the clinical signs and symptoms that are immediately observed. It is therefore convenient to recognize four groups which differ greatly from a clinical standpoint, but each may require tracheotomy, namely, the high spinal, bulbar with 10th nerve involvement, bulbospinal, and encephalitic-bulbar.

Before each type is considered separately, one fact must be mentioned—A tracheotomy mainly accomplishes two purposes: firstly, the bypassing of laryngeal obstruction due to vocal cord paralysis or collapse of the supporting neck muscles and, secondly, the removal of secretions obstructing airflow to the lungs and thereby preventing atelectasis and pneumonitis. A good example is the patient whose color improves immediately after tracheotomy only to die hours later, with essentially normal lungs and bronchi found at autopsy. Many deaths in poliomyelitis are due to the infection involving vital centers and other organs and not necessarily to pulmonary complications, as some clinicians would have us believe.

TYPES OF POLIOMYELITIS

1. *High Spinal Type.*—In this type of poliomyelitis only the spinal cord is involved, without bulbar or encephalitic signs.

Indications for Tracheotomy: A tracheotomy is usually not indicated, since the patient can swallow; and normal tracheal and bronchial secretions are no problem, being handled by the cilia and respiratory movements. Also postural drainage may be very useful in cases in which not too much secretion may occur, as indicated by the patient stating that he "feels" mucus in his airway and is uncomfortable. However, if preexisting pulmonary disease, such as bronchiectasis, is present or if there are signs of an impending encephalitic involvement, as shown by restlessness, semistupor, hypertension, etc., then a tracheotomy may be indicated at once. If bulbar signs should develop, then, of course, the same indications as for the bulbospinal type would hold.

2. *Bulbar Type, with 10th Nerve Involvement.*—In the bulbar type there is cranial nerve palsy (of the 9th, 11th, and 12th cranial nerve) or paralysis involving the 10th nerve, without the toxic signs of encephalitis (prostration, hypertension, cyanosis, etc.), but there usually is severe dysphagia.

Indications for Tracheotomy: Not all cases of this type demand a tracheotomy, especially if conservative measures, such as the Trendelenburg position to accomplish postural drainage and efficient suctioning of the pharynx, are instituted. Even though dysphagia does exist, the cough reflex may be sufficient to keep the trachea and bronchi clear. Self-aspiration by cooperative patients in this group cannot be too highly stressed. If conservative measures fail, as shown by restlessness, inability to sleep, disorientation, low degree of cooperation, duskiness or cyanosis (with or without laboratory confirmation of hypoxia or hypercapnia), then tracheotomy should be done. Also if the 10th nerve involvement includes vocal cord paralysis, indicated by either inability to phonate or stridorous inspiration, a tracheotomy is frequently necessary, since the tussive force is broken if the cords cannot close and the airway obstructed if the cords cannot open.

3. *Bulbospinal Type.*—In this type both cranial and spinal nerves are involved, without encephalitic signs.

Indications for Tracheotomy: The indications are the same as for the pure bulbar 10th nerve type, unless the respiratory muscles (diaphragm, intercostals, and accessories) are also involved (decreasing the vital capacity to 600 to 800 cc. and thereby necessitating a respirator), as well as the bulbar component including the 10th nerve with dysphagia and/or laryngeal paralysis, in which case a tracheotomy is indicated at once.

4. *Encephalitic-Bulbar Type.*—In this type there is diffuse clinical involvement of the cerebrum, with or without involvement of vital centers of the brain stem.

Indications for Tracheotomy: Definite indications for this type are such signs and symptoms as coma or irrational responses, severe hypertension, prostration, various degrees of cyanosis, and unabating fever of 103 to 106 F. (also dysrhythmia of respiratory movements, if the respiratory center is involved).

Although the above descriptions represent four distinct groups of findings, it is not unusual for two or three types to blend, especially in cases in which the encephalitic symptoms are latent and appear after the paralysis has been already evident many hours.

CARE OF THE TRACHEOTOMY

1. *Moisture.*—Moisture must be maintained, either by a vaporizer near the tracheal tube or by a constant moist (sodium chloride or boric acid solution) sponge allowed to loosely rest over the tube.

2. *Suction.*—The catheter should have an open end (not velvet eye) without side holes. One-half of a medicine dropper of sodium chloride solution may be instilled into the tube just prior to suctioning. It is important to insert the catheter down into the bronchi rather than just the length of the tracheal tube. Frequency of suction is determined by presence of secretions. After the first 24 to 48 hours the secretions become heavier for a week or two and then gradually subside.

3. *Mucolytic Agents.*—The use of certain mucolytic agents, such as trypsin or detergents, is frequently indicated to facilitate suctioning.

4. *Supplementary Oxygen.*—Do not permit the catheter to dangle in the tracheal tube, since the direct flow of oxygen into the trachea causes drying of the secretions with crusting and also results in further obstruction to the airway. Use a funnel over the tube or apply a positive pressure attachment.

5. *Cleansing the Tube.*—The inner tube may be changed and cleaned by nurses. The outer tube should be changed by the physician and the frequency determined by him. The outer tube should not be changed for three days after a tracheotomy is made (except in case of exceptional crusting). Subsequently, two or three times a week should be sufficient. Always check the patency of an inner tube by holding it before a light just prior to reinserting. Soaking in fresh peroxide followed by green soap flushes, using a moist cotton applicator, or pulling an opened "four-by-four" sponge through the tube with a hemostat are the most effective means for cleaning. It is not necessary to resterilize the inner tube each time if it is cleaned with green soap. The frequency for cleaning the inner tube will depend on the amount of secretions present. It should be checked at least three times a day. Noisy breathing or whistling sounds indicate partial obstruction in the inner tube and suggest time for cleansing.

DETUBING

The time to remove the tube from a patient varies, of course, with each case. However, if a patient can swallow well, even though he still requires the respirator for breathing, the tube can probably be removed if the tracheobronchial tree and lungs are normal. Persistent bronchial and pulmonary infections may demand leaving the tube in beyond this time. One report states that "the tracheal tube is usually removed by the 16th day, 58 days being the longest period." This statement was made for a series of about 75 tracheotomies.

In long-standing cases it has been our practice to allow the stoma to close down naturally, leaving a small epithelized fistula to persist. The fistula is closed surgically at some later date. If the fistula is retained the patient has most confidence in the fact that, if absolutely necessary, a small catheter could be introduced for suction purposes.

COMMENT

The above outline was prepared originally as a guide for residents and interns. We realize, of course, that there are extremely controversial aspects of tracheotomy in poliomyelitis, but for the orderly management of a poliomyelitis service some routine must be established. Using our four basic groups as a basis for determining the need for tracheotomy in an individual patient, we have simplified the entire problem of indications for tracheotomy. This plan is submitted for possible similar use by other poliomyelitis services.

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TREATMENT FOR CHRONIC SUPPURATIVE OTITIS MEDIA

Report of Ninety-Six Cases Treated Through the Eustachian Tube, with the Aid of the Catheterizing Eustachioscope

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THIS REPORT is the result of a study of 96 routine cases of chronic suppurative otitis media treated through the Eustachian tube with the aid of the catheterizing eustachioscope. (Cholesteatoma cases are not included in this series.)

I have always been of the opinion that the practical approach for the treatment of such cases is through the Eustachian tube. For that purpose in 1925 the catheterizing Eustachioscope was devised. It is an instrument which facilitates the catheterization of the Eustachian tube under direct vision. It is simple to operate and has contributed much to the present knowledge of Eustachian tube gross pathology and function. Eustachian tube pathology was believed to be centered at the isthmus, which is the narrowest portion of the tube; but with the aid of this instrument, I was able to demonstrate that Eustachian tube pathology is at the mouth of the tube.*

A normal-functioning door to the middle ear is essential in treating middle-ear diseases. The mouth of the Eustachian tube must open and close normally, free from mucous plugs, adhesions, and edema.

The advantages in treating cases of chronic suppurative otitis media through the Eustachian tube in preference to the external canal route are as follows:

1. By examination under direct illumination, pathology at the mouth of the tube is more readily detected and more appropriately treated.

2. According to the present method of ear instillations, the patient is placed in the lateral position, with the affected ear up. But in treating through a catheter in the Eustachian tube, the operator can adjust the head of the patient to any position he believes most advantageous for directing the medication to that part of the middle and communicating adjacent pathological areas. The air is easily displaced through the perforated drum. Here the irrigation is done from within out.

3. In cases in which the external canal is filled with granulation tissue arising from the middle ear, medication through the external canal is not practical. When such cases are treated through the Eustachian tube, effectual cures can be accomplished, provided a through-and-through irrigation is obtained.

From the Ear, Nose and Throat Service, Beth David Hospital.

Preliminary report read before the Beth David Hospital Clinical Conference, Nov. 8, 1948, and the Bronx Otological Society, Nov. 22, 1949.

* References 1 to 5.

4. The following simple experiment will clearly demonstrate that the Eustachian-tube approach has far greater merit: Aqueous medication solution, 1.5 cc., is colored with 2 drops of methylene blue. It is then instilled into a suppurative middle ear through the external canal. Dry the external canal thoroughly. Examine within a few hours for stained suppuration. One will find the pus mildly stained; and the staining is of short duration. Using the same ear a few days later and repeating the experiment via the Eustachian-tube approach, one will find, with the same amount of medication used, that the suppuration is deeply stained and that the stain is present for a few days, thus proving the Eustachian-tube route to be more penetrating.

Ninety-six routine cases of chronic suppurative otitis media were treated through the Eustachian tube during a four-year period prior to December, 1951. Eighteen cases had marginal perforations (2 bilateral); 72 cases had central perforations, and 6 cases had one of the external canals filled with granulations. Of these six, three arose from the middle ear through a central perforated drum, one through a marginal perforation, and the other two were undetermined, since a cure was not effected. Of the 18 marginal cases, 12 dry ears were obtained. The drums remained perforated, but the mucous membrane lining, seen through the perforation, was of a normal appearance. No satisfactory results were obtained in six cases. Of the 72 cases with central perforations, 32 cases resulted in the restoration of the drum, 24 cases in dry perforation, and 16 cases were unaffected.

The six cases reported above with granulations in the external canal arising from the middle ear gave the best response. A complete cure with closure of the drum was obtained in three cases. In one case a dry marginal perforation remained devoid of any granulations. No cure was obtained in two cases, for a through-and-through irrigation could not be obtained, since no medication could enter the middle ear. The response to treatment, when effectual, was very rapid. At no time were more than nine treatments necessary to effect a cure.

X-rays of the mastoids and cultures of the suppurations were made in the earlier treatments but were later discarded. X-rays of mastoids (chiefly sclerotic mastoids) of the cured ears showed no changes from those taken prior to treatment. Patients in whom cholesteatoma was suspected were x-rayed for diagnosis. Cultures were later discarded, for the expected results to the specific antibodies and chemotherapy did not always materialize, probably because of mixed infections. I found that a multiple combination of chemotherapy and antibiotics, either in solution or in suspension, is more effective.

MEDICATION

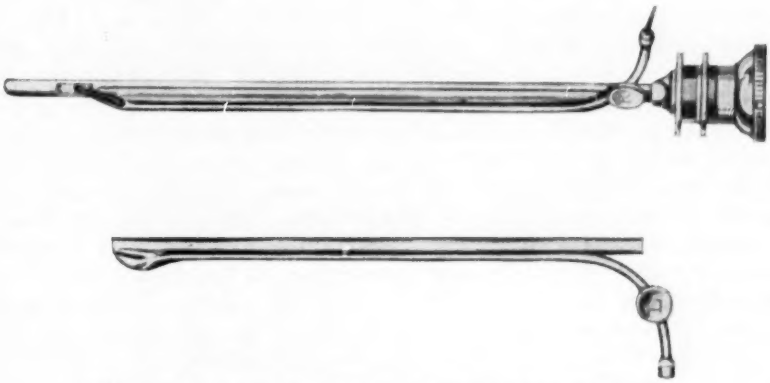
To a vehicle of 1.5 cc. of freshly prepared 30% sulfathiazole sodium solution is added 100,000 units crystalline penicillin G potassium, which is used for the first three treatments, and this is continued if a favorable response is obtained. If not, another antibiotic is substituted for the penicillin, either in suspension or in solution. The quantity is regulated by the consistency of free passage through a No. 4 kidney catheter.

The medication is introduced into the middle ear with the aid of the catheterizing Eustachioscope. The instrument consists of a nasopharyngoscope and two catheter guides, each of which can be mounted on the nasopharyngoscope with the aid of the sleeve. The guides are marked R and L.

The catheter guide R is used in combination with the nasopharyngoscope when the right Eustachian tube is to be catheterized and the guide L in the same manner when the left Eustachian tube is to be catheterized.

DIRECTIONS FOR USE OF THE CATHETERIZING EUSTACHIOSCOPE

To catheterize the right Eustachian tube, the operator holds the eyepiece of the nasopharyngoscope in front of him, with the marking found at the side of the eyepiece toward 9 o'clock. He then mounts the catheter guide R on the nasopharyngoscope. The inlet or handle portion of the catheter guide is also directed toward 9 o'clock. In that position, with the inferior meatus under slight cocainization, the combined instrument is introduced into the right side of the nose along side the inferior turbinate as far as it will go. In that position the orifice of the Eustachian tube will be brought into view. The handle of the catheter guide is turned clock-



PITMAN CATHETERIZING EUSTACHIOSCOPE

wise toward 10 o'clock, and a suitable catheter, after passing through the lumen of the catheter guide, can easily be directed into the Eustachian tube.

To catheterize the left Eustachian tube, the procedure is reversed. The operator holds the eyepiece of the nasopharyngoscope in front of him, with the marking at the side of the eyepiece toward 3 o'clock. The catheter guide marked L is mounted on the nasopharyngoscope so that the handle portion of the guide is directed also toward 3 o'clock. In that position the combined instrument is introduced into the left side of the nose alongside the inferior turbinate as far as it will go. The handle of the guide is turned counterclockwise toward 2 o'clock. A suitable catheter can now be directed into the left Eustachian tube under direct vision.

The attachments have no unnecessary flaps to guide the catheter. There are no other unnecessary parts to complicate a simple instrument. This instrument can be used in all cases.

The catheter used is a 12-in. (30.5 cm.) section of a No. 4 ureteral catheter having a forward outlet. A No. 21 hypodermic needle is attached to the other end. Air is first forced with the aid of a 5-cc. syringe through the catheter to definitely establish a through-and-through circulation before medication is attempted.

SUMMARY

As a result of a study of 96 cases of chronic suppurative otitis media (cholesteatoma not included), the Eustachian-tube route is definitely established as the method of choice for treatment. The medication, procedure, and use of the catheterizing Eustachioscope are fully described.

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RHINOPLASTY AS AN ESTHETIC PROCEDURE

Notes on Artistic Application to Reconstructive Surgery of the Nose

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A TRULY successful rhinoplasty should embody artistic as well as technically sound surgical concepts. It is true that fundamental principles of operative procedure must be observed and must be performed in their proper sequence, but these alone, it is often seen, are not sufficient to obtain the maximum result and the utmost satisfaction.

The nose is the most prominent organ of the face. As such, it plays a predominant role in determining the character as well as the personality of the individual. If a face is pleasing to the beholder, it is usually because that face has a pleasing nose. For centuries physiognomy, the science of determining character and individuality as well as judging the composite personality, has been of consuming interest to many philosophers and poets as well as physicians and surgeons. In ancient Indian writings, in a treatise on the "Arts of Courtship," there is a chapter on the classification of types of women, correlating the size and shape of their noses with their personalities and sensibilities and with their potentialites as successful wives.[1] There have been papers, books, and articles written on "Physiognomy and Genius," "Physiognomy and Speech," and "Physiognomy and Expression," as well as on many other facets of personality as determined by outward characteristics of the face. As Jeremy Collier wrote many years ago: "As the language of the face is universal, so ... ry comprehensive: 'tis the shorthand of the mind, and crowds a great deal ... room."

In every library of any size in the world there is a tremendous store of material on the subject, some still extant from the early 17th century.* And in the face, the largest opportunity for judgment of character has been assigned to the nose. Therefore it becomes of vital moment that in reconstructing the nose, one assign the utmost importance to esthetic principles and artistic concept. It is not sufficient that the operation be learned by rote—an impossible task in any case—since each nose is as different as the person who bears it, but the principles of art and intellectual understanding as well should be applied to the task.

There is an art as well as a science therefore in performing a successful rhinoplasty. The science, which means actually merely the technique of rhinoplasty, has become fairly well standardized in recent years. The concept of some of the facts of the operation has changed and, in some particulars, has undergone basic, fundamental changes as to what is to be accomplished, and in these, I believe, lie sound esthetic principles.

* References 2 to 4.

It would be well to state first the technique used by me and then to follow this with a brief but definite analysis of the artistic dicta of pleasing form and gentle line, which the surgeon should always bear in mind.

Technique of the operation: The vestibular vibrissae are carefully clipped and the nose cleansed. The intranasal area is first anesthetized by the use of small cotton packs or tightly wound applicators sparingly impregnated with a solution of 2% tetracaine (Pontocaine) hydrochloride or with a solution of equal parts of 10% cocaine and 1:1,000 solution of epinephrine. (Although the cocaine-epinephrine solution is more efficient than that of the tetracaine, the dangers attendant upon the use of the former solution must be borne in mind.) These are so placed intranasally that the solution is in intimate contact with the points of emergence of the medial, lateral, and external branches of the anterior ethmoidal nerve along the inner aspect of the dorsum of the nose and the anterior portion of the inferior nasal concha. I also anesthetize the sphenopalatine ganglion immediately posterior to the middle turbinate. Several minutes are allowed to elapse before infiltration of the local anesthetic is begun. Thus there will be little, if any, pain suffered by the patient during the subsequent course of the operation. (The only complaint ordinarily made by the patient is at the moment of the first insertion of the needle at the beginning of the infiltration, and calm reassurance at this point is usually sufficient to allay his fears.) Infiltration is performed by the use of 2% procaine or 1.5% butethamine (Monocaine) hydrochloride, to which a few drops of epinephrine solution (1:1,000) has been added. (The two are about equally suitable for use, although butethamine seems to provide a somewhat more rapid onset of useful anesthesia.) The dorsum is infiltrated sparingly subcutaneously, as well as the base, the tip, and the infraorbital region. At least 10 minutes is allowed to elapse before operation is begun.

The dorsal pyramid, consisting of the nasal bones and the upper lateral cartilages, is elevated through an intranasal incision at the junction of the upper and lower lateral cartilages. Whether the No. 15 blade is used for the entire elevation or the double-edged knives of Joseph and Fomon are used is of little importance as long as the pyramid is uncovered as close above the periosteum as possible and that there remain no subcutaneous adhesions to hamper postoperatively the proper draping of the skin over the newly reconstructed nose, the healing process, or the drawing of the subjacent parts by cicatricial contraction during the succeeding weeks of convalescence.

The nose is transfixed immediately beneath the caudal (inferior) border of the nasal septum and the incision carried back to a point two-thirds of the distance from the tip to the posterior extremity of the cartilaginous septum. The posterior one-third is separated from its attachment to the maxillary spine with scissors. (The incision also may be begun at the caudal edge of the septum, and the septum first transfixed, and the intercartilaginous incision then made to meet it at its anterior end. In either case, it is imperative that the membranous septum between septum and columella be put on the stretch and spared in order to obviate the subsequent contraction of this "rubber" structure, which would then result in an unsightly retraction of the columella.) The saw or the Kazanjian type of rongeur may then be used for the removal of the nasal hump. If the saw is used, the periosteum is incised and raised over the bony portion to be excised on either side of the hump.

After the removal of the bony hump with the saw or saws, the dorsum is trimmed below the bony portion by the use of a short button-end or curved cartilage knife, so that the line of the dorsum is straight and pleasing in its form. If the Kazanjian or the Cottle-Kazanjian rongeur is used, the saw is not necessary; the periosteum need not be raised, and the ensuing line of the dorsum is cleaner in its appearance (Fig. 1). I believe that the rongeur is easier to use, particularly in the removal of a small hump, and that the technique of its use is simpler and more rational, particularly in an operation in which emphasis is placed on cleanness of line. After the use of the hump rongeur, it is usually not necessary to use the button-end knife to trim the cartilaginous dorsum.

At this point, the remaining attachment of the upper lateral cartilages from the septum is severed on either side. It must be borne in mind that should there also

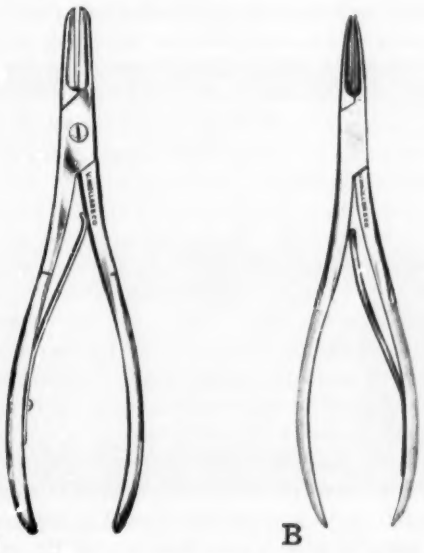

Fig. 1.—(*a*) Kazanjian cutting forceps for removing the nasal hump. (*b*) Cottle-Kazanjian cutting forceps for removing the nasal hump; lighter and smaller than the Kazanjian forceps.

be present a deviation of the nasal septum it is better to perform this step subsequently to the submucous resection of the nasal septum, since if the severance of the attachments between the upper laterals and the septum is done before the submucous resection there will be difficulty in the completion of the submucous resection due to collapse of the intranasal membranes, which no longer are held in their place at the angle between the upper laterals and the septum.

The nose is narrowed by severing the nasal bones from the maxillary processes of the maxilla. An incision is made at the base of the pyriform sinus laterally on either side; the periosteum is elevated in a narrow path on a line extending from the widest point of the pyriform sinus below to the point midway between the inner canthus of the eye and the nasion above. The saw is introduced beneath the periosteum along this line and the nasal bones sawed through until the "mushy" resistance of the nasal mucosa is encountered. The membrane should not in any case be torn.

The nasal bones and their attached upper lateral cartilages are completely separated from the maxillary processes by infracturing, by outfracturing, by the use of a special forceps, such as the Walsham or the Asch, or simply by laterally applied thumb pressure, according to the density of the bone involved. Ordinarily, thumb pressure alone to mobilize the parts is sufficient following the use of the saw.

After this, the lower lateral cartilages are delivered after a lower rim incision has been made between the lower border of the lower lateral cartilage and the vestibular border. (I prefer not to make this incision into the lower alar cartilage itself, believing that in this way opportunity may be provided for rim adhesions and the possible incidence of nicking of the nares, with its subsequent cicatricial contraction.) The alar cartilages are delivered and the lateral and mesial crura severed exactly at their common angle. The mesial crura are delivered, and if their separation is excessive the intervening soft tissue is excised and the mesial crura then sutured at their tip, a mattress suture and straight needles being used. This results in a narrowing of the columella, if this seems indicated, according to the principle of artistic proportion of the columella to the reconstructed nose and, it must again be emphasized, to that of the face. The septum is now trimmed, not routinely excised, by placing the mesial crura at the angle to be desired against the septum and the caudal extremity then trimmed to the proportionate length. This should not be a routine procedure but should be done after the mesial crura have been delivered and sutured together. In this way there will be less danger of excising too great a triangle than if this is done routinely and by rote. The columella is fixed posteriorly to the septum by an absorbable suture, and the lateral crus of the lower lateral cartilage is denuded of its subcutaneous tissue. It is then molded by the breaking of the resilience of its fibers and by the removal of a small segment from its outer subcutaneous surface alone, the upper, lower, and medial borders of the lateral crus being left intact. (Since this step is of the greatest importance in the remolding of the nasal tip, a few statements in this regard will be made in the comment to follow.) The cartilages are molded and replaced, the columella approximated to the septum anteriorly and without tension with one or more absorbable (gut) sutures, the alae narrowed, if this seems indicated, and a fixed splint applied. A sheath of short strips of adhesive (3 by ¼ in. [8 by 0.6 cm.]) is applied as a mold to the nose from the tip to the nasion, each strip being in good approximation with the one beneath it, and one slightly overlapping the one beneath it until the nasofrontal angle is reached. A piece of flannel is cut to shape and then a splint of dental molding wax compound (Stent) or one of shaped tin is applied to keep the nose in fixed position and free of trauma and movement. I prefer to apply a splint cut from a piece of tin sheet metal and covered with adhesive, preferably of flesh color. The Stent splint is excellent, but the light tin splint shaped to conform with the nose is psychologically more acceptable. The latter is also lighter in weight and more comfortable. It may be kept in place by the use of adhesive straps incorporating applicator sticks held together with rubber bands, according to the method of Overholt.[5] A strip of adhesive tape with the wrapped applicator sticks is placed diagonally on each cheek so that a rubber band can be placed between them to hold the metal splint in place over the bridge of the nose. The technique of preparing them is as follows:

> A strip of adhesive tape, the size depending upon the size of the gauze covering the wound (in our case, the metal splint . . .), is made about one by six inches, is stretched out on a table with the gummed surface up. Three fragments of an applicator stick are used, two are cut so

that they are as long as the adhesive strip is wide, and the third is cut ½ inch longer. The longest stick is placed across the adhesive strip 1¼ inches from the end. Each end of the stick extends about ¼ inch beyond the edges of the adhesive strip. The other two sticks are placed parallel and next to the first so that their ends are even with the edge of the adhesive strip. The short end of the adhesive strip is then folded over these applicator sticks. The first stick holds the rubber band while the second and third keep the ends of the straps flat when the rubber band is in place and prevent the strip from folding or doubling up . . .

It will be found that the metal splint or the Stent dressing will be kept easily in place by utilizing two strips of flesh-colored adhesive so prepared. In order for the remolded tip to "set," it is essential that the splint, in the absence of complications, not be moved for five to seven days (Figs. 2 and 3).

The necessity for having clearly in mind artistic concepts of form and line will become apparent in every step of the operation. Even though the reestablishment of a functionally normal nose is the one factor which outshadows all of the others as

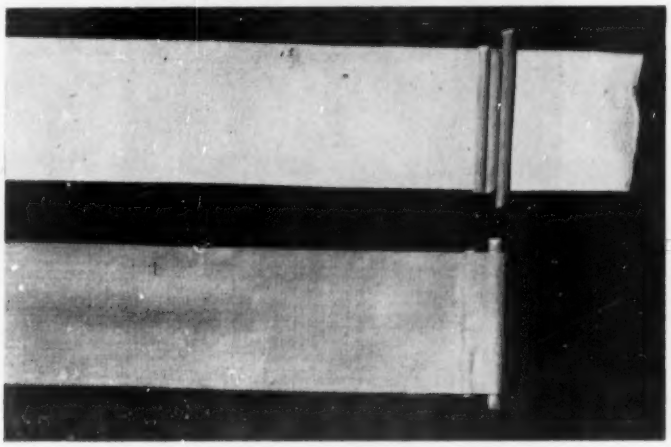

Fig. 2.—Strips of adhesive tape with applicator sticks (front and back views).

an indication for reconstructive surgery of the nose, little mention will be made of this in this paper, since it is, or should be, taken for granted. Besides, Proetz has shown, as have Fomon and others,[6] that a nose grossly deformed externally, whether by a deep saddle or by a long hump, is rarely, if ever, functionally correct (Fig. 4). There is always some difficulty in breathing in these cases—in the hump nose the air currents are too high, and in the saddle nose they are too low.

Though the nose should be so reconstructed in any case that physiologic malfunctions and their correction are constantly kept in mind, still artistic principles should be used so that a pleasing, harmonious nose is the result.

The rhinoplastic surgeon is a sculptor of the living nose and, as such, should follow, insofar as is possible, artistics laws. When the nose is narrowed, the vault will be more pleasing to the surgeon, as well as the patient, if the dorsal crest is not made too sharp, in which case it will present a "hatchet" appearance, or too blunt, in which case it will appear "Negroid" or "flat." The line should be, as in the case of a good piece of sculpture, pleasing and soft.

In the reconstruction of the middle cartilaginous vault, consideration must be made of the fact that functionally the upper laterals may be short, in which case no purpose is served in routinely excising a portion of their lower borders, as otherwise in such a case there may result a slight falling-in of the midportion of the nose. Besides, if the upper laterals are short and are partially excised, there may result a faulty valve mechanism in respiration, since the upper laterals have a definite function in ingress and egress of currents of air. This may easily be shown if the fingers are placed astride the middle cartilaginous vault. At this point, the slightest pressure on the nose will result in difficulty in breathing far out of proportion to the pressure applied. Excision of an already short upper lateral may have the same result.

Fig. 3.—Patient with splint applied to nose.

If the upper laterals are long, they may be partially excised at any time, lest on shortening the nose the airspace be unduly reduced by their added thickness within the nares. I prefer to trim the upper laterals as late as possible in the operation, so that any excess which protrudes at the completion of the operation is excised (no more than is necessary to provide for adequate respiration).

In the reconstruction of the nasal tip lies the greatest opportunity for the surgeon to embody the principles of art and of sculpture, the blending of the esthetic with the scientific. Until recently, this has not been done. Cutting strips off the lower and upper borders and from the extremity of the lateral crura of the lower lateral cartilages, as has been done until recently according to the traditional technique of Joseph, is not artistic in concept and follows no esthetic law of form or line. The sculptor tries to avoid harsh, hard lines in his modeling of a nose. Since it is the function of the rhinoplastic surgeon to duplicate as far as possible the nose of the

talented artist in clay, it would appear that the traditional method heretofore used in rhinoplasty of trimming the borders of the lateral crus of the lower lateral cartilage is not esthetically correct in that more or less sharp edges will always result upon the replacement of the lateral crura and in that these will seem to be sharper than normal under the skin. That this is so can often be seen in a nasal tip reconstructed according to the heretofore traditional technique. It is this trimming of the borders of the lateral crura of the lower lateral cartilages which so often results in a more-or-less "pinched" look.

On the other hand, if the resiliency of the lateral crus is first eliminated and the "spring" of the cartilage is done away with, according to the method first advocated by Fomon and his associates,[7] there will result a cartilage of almost the consistency of putty, and it can then be molded in much the same way in which clay is used by the sculptor in modeling. Only in this manner and only by this method may the form and line and method of the artist be intelligently employed. It cannot be done without destroying the resiliency of the cartilage. The natural nose does not incorporate sharp angles in its tip, nor does the nose of the sculptor. Therefore it

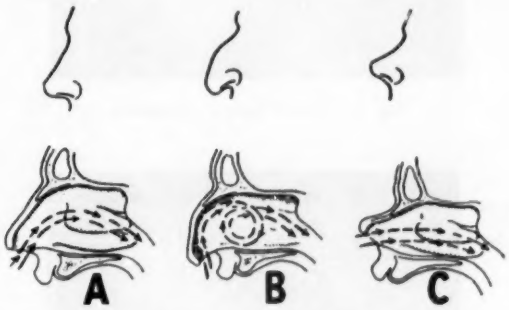

Fig. 4.—Inspiratory current. *A*, normal parabolic curve; *B*, impingement of inspiratory current on dorsum and formation of eddies in case of dependent tip; *C*, flattening of arch and more direct course of current in case of unduly elevated nares.

follows that the rhinoplastic surgeon should strive for the same smooth confluence of lines as the sculptor does. Trimming the borders of the cartilage cannot follow these esthetic lines as well as this method of molding of the tip. If there is too much bulk in a large cartilage, a scaphoid-shaped sliver may be excised from its outer subcutaneous surface, in all cases avoiding the upper, the lower, and the medial margins. By such a method a true harmony of the tip with the remainder of the nose will be achieved.

In summary, attempts have recently been made (Fomon, A. M. Brown, and others) to analyze the artistic criteria which should guide the surgeon in his conception of what is to be done at operation in obtaining the most harmonious synthesis of the lines and form of the nose in regard to that of the face. The face has been divided into thirds vertically and the nose assigned to the middle third. It has been postulated in a full face view that the nose should have the width, at its base, of that distance between the inner canthi of the eyes; that the tip should not lie below a line drawn between the two alae at points joining the alar-labial angles; that the line of the

reconstructed dorsum should form an angle of 30 to 35 degrees with the vertical nasion-anterior maxillary line, and that the columella should be at about 90 to 95 degrees with the upper lip.

These concepts, generally speaking, are valid ones in the average case. But they cannot be combined, necessarily, to form a pleasing nose by using them in routine

Fig. 5.—Patient before rhinoplasty.

Fig. 6.—Patient after rhinoplasty.

fashion. They cannot be used as stereotyped criteria, to be learned as one learns the branches of the maxillary artery. They are subject to endless variations, often minor it is true, but just as often are not maximally pleasing to many types of faces unless accompanied in the surgeon's mind by an artistic sense and an esthetic sensibility. Just as the sculptor never learns facts and figures in molding a beautiful head, so must the surgeon avoid making merely a science of a branch of surgery which does not lend itself to tabulation and routine (Figs. 5 and 6).

SUMMARY AND CONCLUSIONS

A method of rhinoplasty is presented. It is urged that reconstruction of the nose be done with intellectual understanding and artistic appreciation in each step of the procedure and that these concepts are valid and important when they are combined with a sound technique. It is shown that rhinoplasty is not a routine or a technique but an understanding molding of human tissues in sculptured form.

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OSTEOMA OF THE EXTERNAL AUDITORY CANAL

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NEW GROWTH of bone as the result of inflammatory and traumatic conditions is rather common; but in addition to these reparative processes, there is an occasional proliferation of bone without apparent cause or purpose. This latter condition results in the formation of a bony tumor, or osteoma, which eventually causes obstructive and pressure symptoms characteristic of its location. It is well known that the bones of the skull, especially those around the nose and ear, offer the most favorable chances for the development of such tumors.

Osteomas grow slowly, are benign, and are not infrequently multiple. They may be sessile or pedunculated. Microscopically, they vary from dense eburnated bone to soft spongy bone. Often there are no symptoms of their presence, but as they increase in size there may be pressure upon contiguous structures, causing pain and discomfort. They may encroach upon or invade the accessory sinuses, nares, mastoid cells, or ear canals. If near or on the surface, they may cause bulging deformities of the face and head.

The bony tumors of the temporal bone include single and multiple osteomas of the body and surface of the mastoid, the small exostoses of the external ear canal situated close to the tympanic membrane, and the large solitary bony growths which sometimes completely block the external auditory canal. In 1940 Simpson [1] published an exhaustive and comprehensive paper on osteomas of the mastoid in which he reviewed 30 cases previously reported and added 2 of his own. Exostoses of the external canal, which are usually multiple and bilateral, are seen rather frequently. However, they are of clinical interest only in the occasional case with symptoms of threatened obliteration of the lumen of the bony canal. The large solitary osteoma attached by a pedicle to the bony canal wall is an extremely rare condition. The literature on this subject is somewhat confusing, since exostoses of the canal have also been included as osteomas in some of the early reports. Therefore no attempt has been made to determine just how many true osteomas of the ear canal have been reported.

In spite of the rarity of these tumors, Druss and Maybaum [2] in 1942 published a detailed report of three cases and brought the subject up to date by reviewing the literature and appending an extensive bibliography. Since that time, Belal [3] reported the only other case that we were able to find. Since an osteoma in this location is so seldom seen, it is of interest in evaluating this condition to review these four cases briefly before reporting the only case that we have personally observed.

Read before the Otolaryngological Section of the Philadelphia College of Physicians, Philadelphia, Jan. 20, 1954.

REPORT OF CASES FROM THE LITERATURE

CASE 1 (Druss and Maybaum).—A man, 26 years of age, had a history of pain and right ear discharge at age 14. At that time he was advised to have a growth of bone removed from his ear. Discharge had continued off and on, being fetid for many years before he presented himself for treatment on account of frequent episodes of pain. A large osteoma completely filled the right external canal. The left ear was normal in appearance and function. The osteoma attached by a bony pedicle to the anterosuperior canal wall was removed in the course of a radical mastoidectomy. This patient disappeared after discharge from the hospital.

CASE 2 (Druss and Maybaum).—A man, 55 years of age, complained of pain, impaired hearing, and noises in the right ear of seven weeks' duration. He had no previous history of ear symptoms. A hard bony mass with a small amount of thin purulent discharge almost completely blocked the right ear canal. There was a conductive type of hearing impairment with approximately a 40-db. loss. The left ear was normal. A modified radical mastoidectomy was necessary to remove the osteoma, which was attached to the anterior canal wall by fibrous tissue and occupied a depression in the posterior wall of the canal. The tympanic membrane was intact. The pathologist's diagnosis was ossifying fibroma. Six weeks after operation, the hearing loss was only 10 db. below normal.

CASE 3 (Druss and Maybaum).—A man, 37 years of age, gave a history of fullness in the right ear of about one year's duration. A stony hard mass almost completely occluded the right external ear canal. There was a hearing loss of 45 to 50 db. in the right ear. The left ear was normal in appearance and function. The osteoma, which was found to be attached to the anterior and superior canal walls by a bony pedicle, was exposed and removed by means of an endaural modified radical mastoidectomy. The ear drum was intact. The pathologist's diagnosis was a typical bone tumor.

CASE 4 (Belal).—A man, 30 years of age, complained of right-sided deafness and a hard mass protruding from his right ear. There was no pain, headache, or tinnitus. The mass was firm and fixed; the covering epithelium was smooth and bathed in mucopurulent discharge. Hearing tests (kind not specified) showed complete loss of air conduction but normal bone conduction in the affected ear. The other ear was normal in appearance and function. The tumor, attached to the anterosuperior portion of the bony canal wall, was removed through a postauricular incision after a section of the posterior canal wall and neighboring mastoid cortex had been taken away. It consisted of eburnated bone. There was a central perforation of the drum that healed in one month, and two weeks later the hearing was normal. The tumor was 17 by 12 mm., the third dimension not being given. This case is of particular interest because it is probably the largest reported osteoma of the ear canal and also because the author reported complete loss of hearing by air conduction before operation and normal hearing six weeks following operation.

REPORT OF A PERSONALLY OBSERVED CASE

Mrs. E. H., 51 years of age, was complaining of fleeting sharp pains in her left ear when first seen by us. She said that as long as she could remember she had been unable to hear with her left ear but that there had been no other ear symptoms until approximately five weeks before consulting us. She recalled no symptoms of an upper respiratory infection at the onset of ear symptoms or since that time, nor was she ever conscious of any ear discharge. About the first week of October, 1951, she began to notice transient sharp pains in her left ear. These pains gradually became worse, and so on Oct. 25 she consulted her family physician, who gave her a series of seven penicillin injections. Her discomfort in and around the left ear continued with occasional dizziness and a tendency to fall to the left. After being kept awake one night with pain, dizziness, and nausea, she was advised to consult us. However, her severe symptoms promptly subsided, so she did not come to our office until three days later.

When we examined her, she was comparatively comfortable, having only an occasional fleeting sharp pain in and behind her left ear. She had no elevation of temperature and said that she had had no fever at any time. There was no spontaneous nystagmus, but she did have a tendency to fall to the left side. There was no evidence of infection or other pathology of the nose and throat. The right ear was normal in appearance and function. The left ear canal was completely occluded by a hard bony mass bathed in a scant fetid discharge, and there was some

tenderness over the mastoid antrum. She was unable to hear a police whistle or the sound of a shouted voice a few inches from her left ear when the right ear was masked with a Bárány noise box. Roentgen examination showed poor aeration and lack of definition of the normal cellular pattern of the left mastoid, while the right appeared to be normal. There was also noted an abnormal shadow of bone density situated in the left external canal. A diagnosis of suppurative mastoiditis, left, with osteoma of the external auditory canal was made and operation advised.

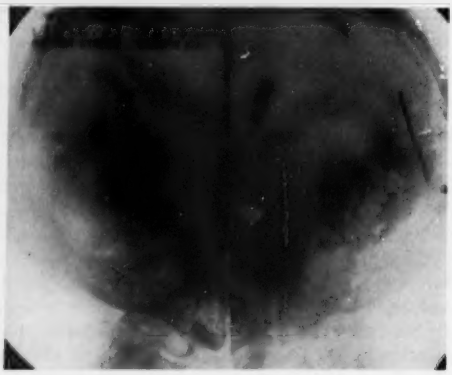

Fig. 1.—Roentgenograms of mastoids showing the extensive cellular destruction of the left as compared with the normal right mastoid.

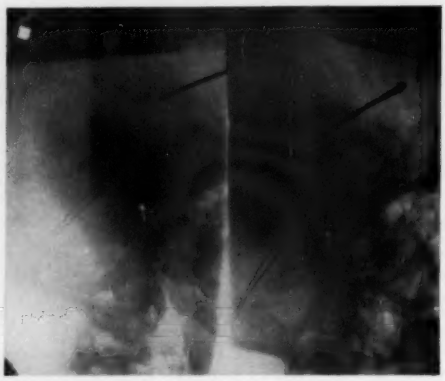

Fig. 2.—Roentgenograms of the left mastoid showing osteoma in the external canal, with the jaws closed in one view and open in the other.

She was admitted to the Cooper Hospital Nov. 19, 1951. The urine, blood cell count, and blood chemistry studies revealed nothing abnormal. Physical examination was negative except for the left ear and mastoid. The highest temperature recorded before operation was 99 F. On Nov. 20 the mastoid and osteoma were exposed through a postauricular incision. A small collection of subperiosteal pus and a necrotic area of the bony cortex were found in the region of the mastoid antrum. After a portion of the posterior canal wall and neighboring mastoid cortex was removed, the osteoma was pried loose from its attachment to the anterosuperior canal wall. After the osteoma was removed, the tympanic membrane was seen to be almost completely destroyed and the middle ear filled with fetid pus. We decided that a radical mastoidectomy was justified. In carrying out this procedure, we encountered extensive bone involvement,

necessitating another incision at right angles to the first for adequate exposure. Recovery from the operation was uneventful. The patient was discharged from the hospital Dec. 1. Culture of pus from the mastoid yielded Staphylococcus albus.

When this patient was recently examined, the ear was dry. The audiogram showed only 30 to 45 db. loss of hearing for the lower frequencies, but for frequencies above 1024 cps the loss was considerably greater. She was able to repeat words spoken with conversational voice

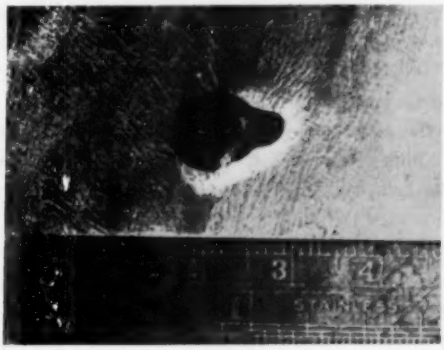

Fig. 3.—Gross appearance of osteoma in the largest diameter. The rule shows metric measurement.

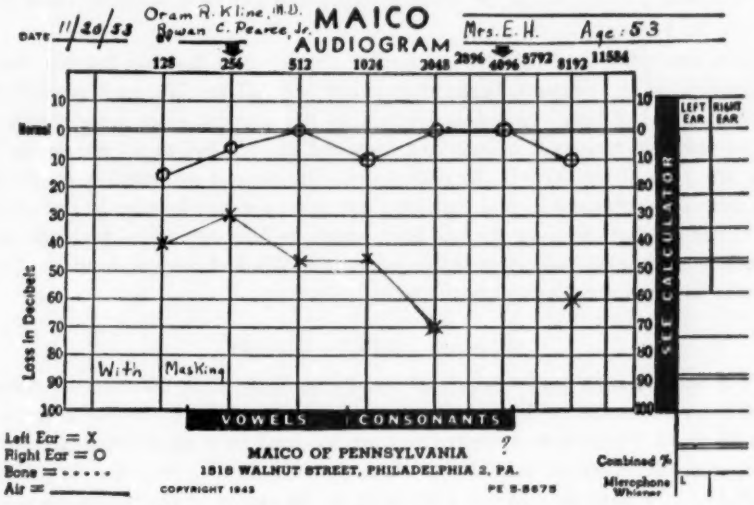

Fig. 4.—Audiogram two years after operation.

intensity 1 in. (3 cm.) from her left ear with the same masking of her right ear as before operation, when she could not hear the sound of a shouted voice at the same distance.

COMMENT

This case presents certain features that merit discussion. How can one rationalize the history with the pathology found at operation? Here is a woman, 51 years of age, who states that she had never been able to hear with her left ear but does not

recall ever having had any other ear symptoms until approximately seven weeks before operation, and during that time there was no fever or ear discharge. Her symptoms were limited to fleeting sharp pains in and behind the ear, with the exception of a 24-hour period a few days before operation, during which time she had continuous severe pain accompanied by vertigo and nausea. The pathology found included blocking of the external canal by a bony tumor covered with thin skin moistened by seepage of pus, widespread infection of the middle ear and mastoid with destruction of the tympanic membrane, and bone necrosis to the extent of perforation of the cortex.

There was no history, symptoms, or signs of an upper respiratory infection preceding or during the acute ear symptoms, and the only organism cultured from the pus was Staph. albus. Therefore it seems logical to assume that the osteoma caused erosion of the tympanic membrane, permitting the Staph. albus, normally found on the skin, to invade the middle ear and mastoid.

Staphylococci seem to have a special predilection for bone. Although Staphylococcus aureus is more virulent and more frequently found in bone infection, Staph. albus can also cause bone necrosis. However, owing to its lesser virulence, the disease usually progresses more insidiously and causes less systemic reaction. Just when the middle ear in this case was invaded is not known, but no doubt it was some time before acute symptoms occurred. It will be recalled that Belal's patient had a central perforation of the drum at operation, although he complained of no pain or headache.

It is also rather unusual that our patient should insist that her hearing was no worse after acute symptoms began than it had been all her life. Is it possible that the ear canal had been completely blocked by the osteoma since early childhood? In the cases we reviewed, fullness and impaired hearing had been present for less than one year, if we exclude the patient with the chronic suppurative mastoiditis. We were also surprised to find that after operation she had hearing in the affected ear by air conduction, since she had been unable to hear any sound with the police whistle and shouted voice test before operation. We had always considered such cases to have irreversible total deafness.

SUMMARY AND CONCLUSIONS

The solitary bony tumor which eventually tends to block the external auditory canal is a rare condition. We found reports of only four cases in the literature of the past 15 years. These were reviewed and are followed by a more detailed report of our personally observed case. In all these patients the osteoma was found to be attached to the anterior-superior bony canal in the region of the tympanosquamous suture. From this point of origin it grew toward the drum and toward the external orifice as it increased in size. The tumor apparently is slow-growing and usually has existed for years before the patient is aware of its presence.

If one of these osseous growths is found by the otologist, how should the condition be managed? Even a fairly large osteoma does not demand surgical intervention when it is not causing symptoms. However, the patient should be advised that reevaluation will be necessary whenever there is impaired hearing, persistent fullness of the ear, or pain. If these symptoms do not promptly respond to treatment,

removal of the growth is indicated. When middle-ear disease is suspected and the osteoma interferes with drainage and visualization of the drum, surgical intervention is imperative for intelligent and effective treatment.

The ease with which the tumor can be removed depends upon its size and the width of its pedicle. If it does not completely obstruct the canal and the pedicle is small, removal through the canal is feasible. When the canal is completely obstructed, it is necessary to remove a portion of the mastoid cortex and posterior canal wall. After the osteoma is completely visualized in this manner, it can easily be removed without fear of injurying the drum and structures of the middle ear.

514 Cooper St.

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Case Reports

PNEUMOCOCCIC MENINGITIS FOLLOWING AN UNUSUAL ACCIDENT

Report of a Case with Recovery

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AND
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URBANA, ILL.

PNEUMOCOCCIC meningitis is still a serious illness in children; however, when adequate treatment is started early in the disease a favorable outcome should be expected. Schwemlein and associates [1] demonstrated measurable amounts of penicillin in the spinal fluid of all patients receiving 20,000,000 or more units of penicillin by continuous drip over a 24-hour period. Following this, it was shown that when 1,000,000 units of penicillin were given intramuscularly at two-hour intervals the spinal fluid concentrations were between 0.08 and 1.25 units per cubic centimeter eight hours after treatment was begun.[2] Ruegsegger,[3] in an analysis of 637 cases, noted that pneumococcic meningitis secondary to infections within the head or injuries to the head had been more amenable to treatment than that which was secondary to pneumonia. The case presented here demonstrates an unusual type of head injury. The child developed pneumococcic meningitis following the injury and made an uneventful recovery from both.

REPORT OF CASE

S. M., a 5-year-old white boy, was admitted to our hospital on April 5, 1953, at 10:00 a. m., in a semicomatose condition. The parents gave a history of the child's falling on April 4 at approximately 10:30 a. m. and ramming a splinter from a cedar shingle into the right nostril. He was taken to a physician, and a splinter 3 to 3½ in. (7.6 to 9 cm.) in length was removed, with the help of a clamp, after considerable effort. Following this procedure there was moderately severe bleeding from both nostrils. He was seen at 4:15 p. m. on April 4 in our ear, nose, and throat department. He had vomited seven or eight times since 12:00 a. m., and the mother had noted an ecchymotic area at the medial aspect of the left orbit.

Physical examination revealed a well-developed, well-nourished, cooperative child of 5 years who did not appear acutely ill. The temperature was 99.4 F. A slight ecchymosis of the medial aspect of the left orbit was noted. There was no active hemorrhage from either nostril, and an area of penetration was noted through the anterior tip of the right inferior turbinate. The antra were clear, and there was no postnasal discharge. The patient was given 450,000 units of penicillin and allowed to go home.

During the night his temperature went to 104 F., and he became very uncooperative and would cry out. He would occasionally grasp at his testicles and penis and had frequency of urination.

Physical examination at 10:00 a. m. on April 5 revealed an acutely ill semicomatose patient. The temperature was 104.5 F. The blood pressure was 120/80. There was crusted blood in both nostrils and ecchymosis about the medial portion of the left orbit. The pupils were equal and reacted to light and in accommodation. The throat was moderately injected. The heart,

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lungs, abdomen, and genitalia were normal. Abdominal and cremasteric reflexes were not obtained. The tricers, biceps, patellar, and Achilles' reflexes were hyperactive and equal. The Babinski reflex was negative. There was moderate nuchal rigidity.

Lumbar puncture revealed a cloudy fluid that flowed freely. The total cell count was 1,760, of which 60% were polymorphonuclear leucocytes and 40% were lymphocytes. The total spinal fluid protein was 376 mg. per 100 cc., and the sugar was 10 mg. per 100 cc. Direct smear showed the presence of bacteria with the characteristics of Diplococcus pneumoniae. Cultures later proved this to be a Type VI pneumococcus that was sensitive to oxytetracycline (Terramycin), penicillin, erythromycin (Ilotycin), sulfisoxazole (Gantrisin), and streptomycin but resistant to chlortetracycline (Aureomycin) and chloramphenicol (Chloromycetin).

The hemoglobin value was 11.8 gm. per 100 cc.; the erythrocyte count was 4,000,000, and the leucocyte count, 31,400, with 85% neutrophiles, 4% stab cells, 9% lymphocytes, 1% monocytes, and 1% eosinophiles.

Urinalysis showed a 1 + reaction for albumin and 10 to 15 red blood cells per high-power field. X-rays of the sinuses and skull on April 7 showed no evidence of fracture or air within the calvarium.

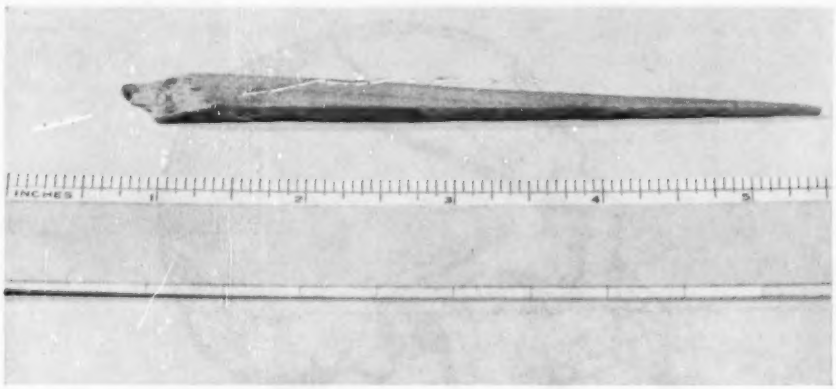

Fig. 1.—Splinter withdrawn from child's nose.

Treatment consisted of crystalline penicillin, 2,000,000 units every two hours, and subcutaneously administered sulfadiazine sodium. On April 6, the patient was rational and ate well. His temperature dropped to 99 F. on April 7, and during the remainder of his hospital stay it did not go above 100.6 F. During his 14 days in the hospital he received a total of 146,000,000 units of penicillin; 3 gm. of subcutaneously administered sulfadiazine sodium; 5 gm. of sulfadiazine, sulfathiazole, and sodium citrate (Citrasulfas, Upjohn), and 29 gm. of sulfisoxazole (Gantrisin).

The spinal fluid examination on April 6 showed a protein value of 90 mg. per 100 cc.; lymphocytes, 12; polymorphonuclear leucocytes, 3, and sugar, 66 mg. per 100 cc. Spinal fluid culture was negative. The patient was discharged from the hospital on April 16, apparently cured.

The child was next seen on April 20 and 22. On both occasions he had a slight elevation of temperature. Physical examination showed the deep tendon reflexes to be hyperactive. His sense of smell was normal.

On April 29 he developed rubeola, from which he made an uneventful recovery.

On May 19 he complained of his nose bothering him, and his parents observed a piece of wood protruding from his right nostril. He was brought to our ear, nose, and throat department, where a wooden stick measuring 12 cm. (Fig. 1) was gently removed with the fingers. It was firmly in place; however, very little pull was required for its removal.

Since then the patient has had no difficulty and has been healthy in all respects.

COMMENT

Figure 2 is our concept of the area penetrated by the splinter. The length of the foreign body and the angle at which it was withdrawn indicated that it had rested in this position. The child was extremely fortunate to have no permanent damage from this injury. Our failure to know that the splinter was present probably was fortunate. Had we realized it was there, some attempt might have been made to remove it earlier, and the final results possibly would not have been as excellent as they were.

The pneumococcic meningitis which followed the injury within a few hours gave all the indications of a fulminating infection. The response to the large doses of penicillin and the sulfonamides substantiates many previous observations.* This child tolerated the large doses; however, as a large volume of fluid was required to dissolve the penicillin and the point of injection was frequently painful for several days, care was exercised in selecting areas for intramuscular injections. The nurses

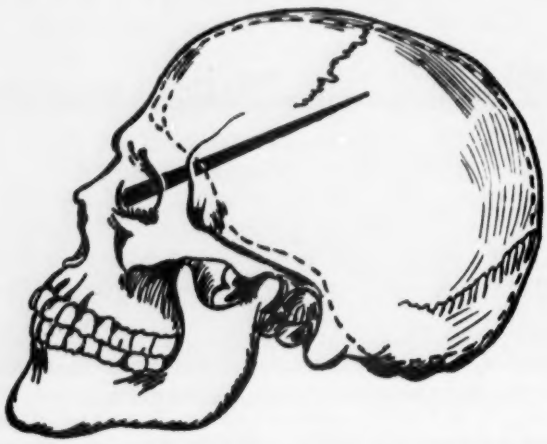

Fig. 2.—Area penetrated by splinter in head injury.

were instructed to distribute the injections over the buttocks, the anterior aspects of the thighs, and the deltoid and tricep areas. By spacing them judiciously, no serious indurations occurred.

SUMMARY AND CONCLUSIONS

A case report is given of a child who developed pneumococcic meningitis following the accidental penetration of a splinter through his nose into the calvarium. Six weeks later a foreign body presented itself at the right naris and was removed easily with the fingers.

This case is presented to further substantiate the excellent results in the treatment of pneumococcic meningitis with sulfonamides and large doses of penicillin. The fortunate outcome in regard to the extrusion of the foreign body would indicate that in case of similar accidents conservative treatment might be preferable to any radical procedure to remove the foreign body.

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PYLORIC STENOSIS AS A SEQUELA OF ALKALI INGESTION

Review and Report of a Case

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THE VARIOUS sequelae of alkali ingestion upon the mouth, pharynx, and especially the esophagus are well known, well described, and widely taught.* The most important of such disorders are esophageal stenosis and stricture. These entities are universally anticipated following acute alkali burns and are treated by various means.† Recently the use of wider surgical procedures in the thorax has become practical in the treatment of these conditions to augment other means.[4] The active prophylaxis of these sequelae by means of early vigorous dilations is being revived.[4] For many years attempts to reduce the incidence of these burns by legislative control over the sale of alkalies have been made by members of the profession.[5]

Such widespread information and study are not the case when the problem of alkali ingestion and its sequelae in the stomach is considered. In fact, most textbooks of otolaryngology fail to mention any gastric sequelae of alkali ingestion.‡ The commonest such sequelae are gastric and pyloric stenosis. I have been able to gather only three reports in the recent American literature dealing with cases of pyloric stenosis following alkali ingestion. Gradman and associates [6] described one case of pyloric stenosis following lye ingestion, without esophageal stricture. Vinson and Hartman [7] described one case of pyloric stenosis, coexisting with severe esophageal stricture, following lye ingestion. Bolstad [8] described two cases of complete pyloric obstruction, with esophageal stricture, one following lye ingestion and the other following alkaline drain-cleaner ingestion.

This is a report of an additional case of pyloric stenosis, with esophageal stricture, resulting from the ingestion of an alkaline caustic.

REPORT OF CASE

H. D. (No. 41,575), a 27-year-old white married woman, was admitted to the United States Naval Hospital, Portsmouth, N. H., on Aug. 13, 1952. Two and one-half hours previously she had ingested, with suicidal intent, approximately 30 cc. of a commercial drain cleaner. The

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The opinions and views set forth in this article are those of the author and are not to be construed as reflecting the policies of the Navy Department.

* References 1 to 3.

† References 1 to 4.

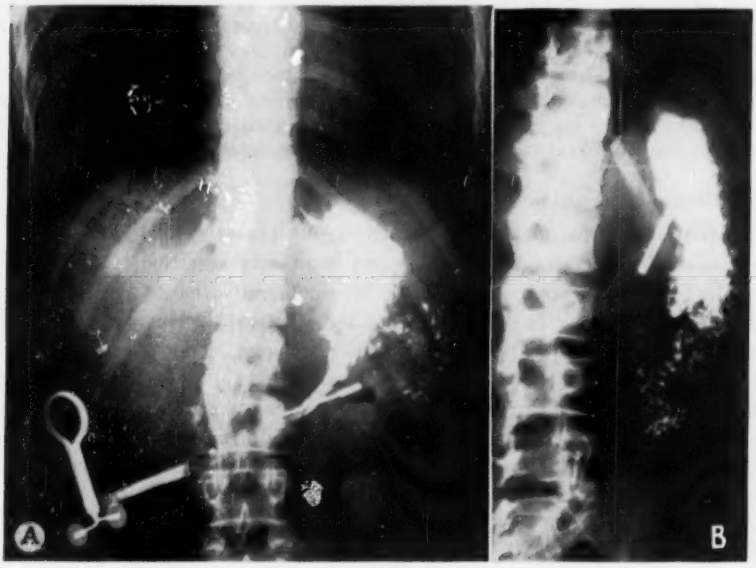
‡ References 1 to 3.

manufacturers of this product state that it consists predominately of sodium hydroxide.§ After being given emetics at home, whereby bloody emesis was produced, the patient was brought 90 miles to the hospital.

On admission, the patient complained of midline anterior chest pain, crampy abdominal pain, and inability to swallow. Examination revealed the temperature to be 98.4 F.; pulse, 88, and blood pressure, 100/76. The patient was vomiting blood-stained gastric juice occasionally. There were burns of the mucosa of the lips, tongue, and pharynx. There was no abdominal tenderness, and bowel sounds were normal. The remainder of the examination was normal.

Past History.—This woman had attempted suicide previously in 1947 by ingesting barbiturate capsules and had threatened suicide frequently. Her life had been marked by a broken home in childhood and two unsuccessful marriages. The past medical history was noncontributory.

Course.—The patient was treated expectantly with sedation, analgesics, antibiotics, and parenteral nutrition until by the 10th day she was able to swallow liquids easily and felt mod-



Roentgenograms: (*A*) Dec. 3, 1952; (*B*) March 9, 1953. Both show opaque media in the stomach and jejunum but none in the pylorus or duodenum.

erately well. In the ensuing 20 days, however, she slowly but steadily grew worse, with decreasing ability to swallow and then increasing upper abdominal pain. On the 28th day esophagoscopy revealed healing burns of the mucosa of the hypopharynx and esophagus. The instrument could not be passed beyond a point 20 cm. from the incisor teeth because of constriction of the lumen. In the next six days the patient's condition worsened; she lost weight rapidly, could not swallow, had increasing abdominal pain and distention, and had waves of pain across the upper abdomen from the right to the left side. On the 34th day the abdomen was explored via an upper right rectus incision. The stomach was dilated and found to be filled with about 1,500 cc. of foul fluid and old food. A Janeway gastrostomy flap was fashioned. On exploring the interior of the stomach, the pylorus was found to be completely obstructed by a concentric constriction at its entrance and in it. It was not possible to pass a tiny probe through the pylorus. A small area of granulations was present at the edge of the constriction. The remain-

§ Personal communication of the author.

der of the stomach was normal grossly. Because of the obstruction, an anterior antiperistaltic gastrojejunostomy was performed, and the gastrostomy was then completed. Esophagoscopy at the same time was no different than previously, but a 12 F. stomach tube was passed into the stomach and left. Attempts to dilate the esophagus through the esophagoscope were only partially successful and tediously slow. Consequently retrograde bougienage was instituted. The patient was able to leave the hospital on the 132d day, being able to feed herself entirely by mouth and showing steady progress in dilatations. She has not used the gastrostomy for feeding since.

Two months later the patient was readmitted for four weeks for a period of regular dilatations and diet regulation. Upon discharge then (some eight months after the injury) the esophagus was dilated to a 24 F. bougie, and the patient was gaining weight. She then left my care. When last heard from in September, 1953 (13 months after the injury), she was using a 34 F. retrograde bougie and felt well.

Studies.—An x-ray report of Dec. 3, 1952 (112 days after injury) read in part: "At no time during fluoroscopy or later did any amount of the opaque medium pass through the pylorus." An x-ray report of March 9, 1953 (208 days after injury) read in part: "At no time is there any filling of the distal portion of the stomach, and, of course, no passage through the pylorus is demonstrated." The Figure represents x-rays taken on these two occasions, and each shows the failure of the barium mixture to pass through the pylorus. Each also shows the mixture passing easily through the gastrojejunostomy stoma. A gastric analysis performed 210 days after injury showed normal free acid after histamine stimulation.

COMMENT

It would seem that alkali ingestion commonly causes esophageal stricture and rarely causes pyloric stenosis. On the other hand, acid ingestion is commonly followed by pyloric stenosis and not so often by esophageal stricture.‖ Possible factors in this difference may be (1) neutralization of alkali in the acid gastric juice, especially weaker alkali [10]; (2) relatively noncaustic action of acid upon the squamous epithelium of the esophagus, compared with the universally caustic action of strong alkali [12]; (3) more immediate severe pain caused by strong alkali ingestion, making further swallowing impossible after one or two gulps, and (4) damaging effects of dilute acids in the stomach, compared with dilute alkalies.[12]

SUMMARY

A case of pyloric stenosis, following the ingestion of a strong alkali and present concomitantly with esophageal stricture, is reported. Four previously reported cases are reviewed. Some possible reasons for the rarity of this lesion are mentioned.

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DEAFNESS AND CLEIDOCRANIAL DYSOSTOSIS

PERK LEE DAVIS, M.D.
PAOLI, PA.

DEAFNESS is disturbing at any age. Its relief is not always obtained by operation or aids. Its causes are manifold. One interesting congenital disturbance which produces progressive deafness as maturing occurs is cleidocranial dysostosis. The following case is reported because it exemplifies that disturbances in cranial bone growth should be considered in progressive deafness in the young adult.

REPORT OF A CASE

A white man, aged 24, consulted the Davis Clinic, Paoli Medical Center, because of deafness. He remarked that impaired hearing began at the age of 14 and became progressively worse until he was graduated from college, at the age of 22. While he was talking, it was observed

Fig. 1.—The patient in a normal position.

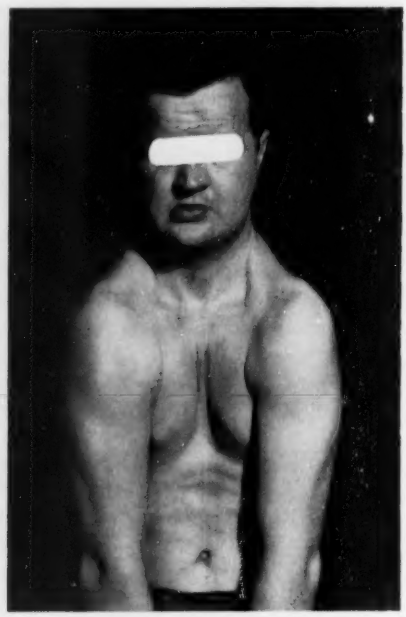


Fig. 2.—The patient in a position in which his shoulders are brought under his chin.

From the Davis Clinic, Paoli Medical Center.

by the examiner that there was considerable facial asymmetry. The patient was asked if he could bring his shoulder under his chin. He was most surprised by this question, stating that he had never tried it. He was asked to try and was surprised when he accomplished this feat (Figs. 1 and 2). He remarked, when it was explained that the examination would most likely show the absence of the clavicles, "Perhaps that is the reason I was such a good wrestler in college. I could wiggle out of almost any hold." Examination and x-ray films revealed skull asymmetry, differences in the greater wings of the sphenoid bone and in the right and left temporal and parietal bones, plus an absence of the clavicles. Bone-conduction hearing aids have given a modicum of improvement. The patient has been observed over a period of seven years, during which time he married. His son, born recently, has the same defect and, in addition, has synostosis of the upper cervical vertebrae—Klippel-Feil syndrome.

COMMENT

Cleidocranial dysostosis may affect the entire skeleton to a greater or lesser degree. It is a congenital abnormality involving the bones of membranous origin. The pathognomonic features are found in the skull, clavicle, and pelvis. The eyes are widely spaced (hypertelorism) ; the nose is depressed at the bridge ; the face is small, and the teeth show faulty eruption. The abnormality is discovered usually during the course of an examination for something else, as it was in this case.

SUMMARY

A case of cleidocranial dysostosis is reported.

Congenital abnormalities of growth of the cranial bones may be a cause of deafness.

Clinical Notes, New Instruments and Techniques

A SET OF INSTRUMENTS DESIGNED FOR STRIPPING THE VOCAL CORDS

JOHN M. LORÉ Jr., M.D.
NEW YORK

Stripping of the vocal cords was used by Loré * for taking biopsy specimens and in the treatment of benign lesions and bowing of the cords. Subsequent experience has also indicated its value in the treatment of unilateral or bilateral cord paralysis. The purpose of this presentation is to describe the set of instruments † used for stripping the vocal cords.

DESCRIPTION OF INSTRUMENTS AND TECHNIQUE EMPLOYED

The original instrument that was designed by Loré ‡ for the operation was based on a modification of the Imperatori subglottic forceps. The stripping instruments are in two sets: one for adults and one for children. In each set there are two types: one for the right and one for the left vocal cord. Each instrument has two blades: one hinged and one fixed. The blades are oblong in shape and concave on the inside. The cutting surfaces are straight. The anterior two-thirds of the cutting surface is sharp, and the posterior one-third is dull. The blades for the adult are 9 mm. in length, and those for the child[2] are 6 mm. in length. They are made in such a manner that the hinged blade is always in juxtaposition with the glottic opening (Figure, *1* to *4*). This arrangement permits an unobstructed view of the free edge of the vocal cord during the initial phase of the operation. Accordingly, an injury to the ventricular band is less likely to occur.

The technique employed in the application of the blades is the same as originally described by Loré [2]: "The blades are then opened (Figure, *1*), and the growth and subjacent cord are engaged gently. Slight traction is then made toward the free edge until the growth itself is felt in the forceps. Then the forceps is closed tighter (Figure, *2*). At this stage it is important to visualize the cord to make sure too much is not being removed. If too much tissue is engaged, the forceps is opened a little until the proper amount of tissue is included in it. Then the growth is stripped from the cord (Figure, *3* and *4*). Gross examination of the growth will show it to have two thin small strips of cord tissue attached to it."

If stripping of both cords is required, six to eight weeks should elapse before the opposite vocal cord is stripped.

If the cords are bowed, owing to either a prominent vocal process of the arytenoid cartilage or to paralysis of the external branch of the superior laryngeal nerve, the tip of the vocal process is removed during the stripping procedure. In the treatment of bilateral abductor cord paralysis, the removal of the vocal process increases the glottic airway and improves the voice.

From the Head and Neck Service, Department of Surgery, Saint Clare's Hospital.

* References 1 and 2.

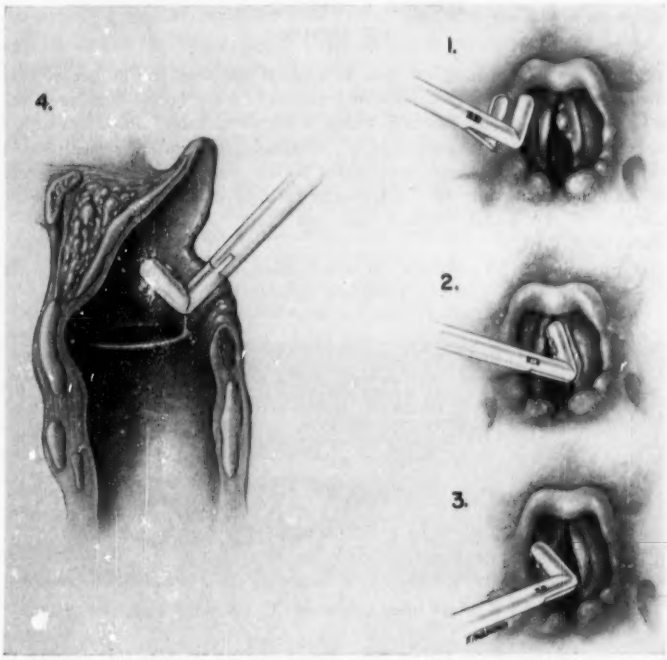
† Manufactured by George P. Pilling & Sons, Philadelphia.

‡ References 1 and 2.

SUMMARY

Stripping of the vocal cords is a relatively simple operation and may be used to obtain a biopsy specimen; to remove diseased tissue; to correct bowing of the vocal cords, and to treat unilateral or bilateral cord paralysis due to injury of either the recurrent laryngeal nerve or the external branch of the superior laryngeal nerve.

A set of instruments designed for stripping the vocal cords is described.



Stripping a vocal cord: *1.* The instrument is inserted with the hinged blade open and parallel to the free edge of the cord (supraglottic view). *2.* The hinged blade is placed parallel to the cord, and the blades are approximated. The stripping maneuver is begun anteriorly (supraglottic view). *3.* Supraglottic view of the straight free edge of the cord after stripping. *4.* Lateral view of the straight free edge of the cord after stripping.

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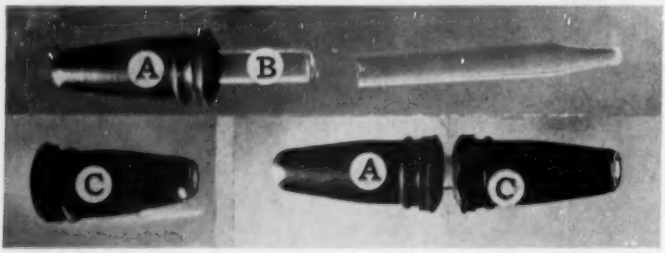
PRESSURE ALTERATION OF THE EXTERNAL AUDITORY CANAL

BERNARD S. POGOREL, M.D.
LOS ANGELES

In the present era of great progress, both in surgical technique and in specific chemical and biological therapy, certain older modalities of treatment have properly been replaced by more effective methods; certain others, however, retain a degree of value, which is worth salvaging.

In certain ear conditions, pneumomassage is a valuable adjunct to other therapeutic measures. A more correct, though cumbersome, designation is "air condensation and air rarefaction" of the external auditory canal, since the method may be applied in cases of drum perforation, in which "massage" of the drum obviously does not occur. A simpler designation might be "pressure alteration."

A list and discussion of the conditions in which pressure alteration of the external auditory canal may be useful is to be found in Politzer's textbook.[1]



A, distal bulb; *B*, glass tube, shortened; *C*, proximal bulb, perforated.

It is my purpose in this article to describe a simple method whereby the principle of pressure alteration may be applied by the patient himself, so that, if the physician desires, the treatment can be carried out as an ancillary measure, several times a day, at home.

The ordinary, straight medicine dropper, with a firm rubber bulb of 1.5 cc. capacity, is used. The distal segment of the glass tube is removed with an ampule file and discarded; the remaining glass segment, which should be about 1.5 cm. in length, is left inserted into the bulb. A second bulb, the tip of which has been cut off, is then fitted to the free end of the glass tube and slid up on the latter until the two bulbs are touching. See illustration. The cut bulb, when inserted into the external canal, assures an air-tight fit.

The maximum pressure reduction which can be obtained with this apparatus, in an ear canal leading to an intact drum, may be calculated as follows:

The average volume of the adult canal, V_c, is 1.5 cc. The volume of the distal bulb, V_d, and of the proximal bulb, V_p, is 1.5 cc. for each. Allow P to symbolize the final pressure in atmospheres. Then, if the distal bulb is completely collapsed between the thumb and index finger, the proximal bulb inserted into the canal, and the distal bulb allowed to expand fully,

$$P = \frac{V_c + V_p}{V_c + V_p + V_d}$$

$$P = \frac{1.5 + 1.5}{1.5 + 1.5 + 1.5} = \frac{2}{3}$$ atmosphere, or 506.6 mm. of mercury, thus representing a pressure reduction of 253.4 mm. of mercury.

1. Politzer, A.: A Text-Book of the Diseases of the Ear, edited by M. J. Ballin and C. L. Heller, London, Baillière, Tindall & Cox, 1909, p. 146.

The usual Siegle otoscope has a bulb with a volume of 38 cc., a connecting tube about 8 cc., and the ear piece itself about 9 cc. Such an otoscope will reduce the pressure to approximately one-third of an atmosphere, or about twice as much as the apparatus described above. Similarly calculated, should the distal bulb be replaced with one of ¼ oz. (7.5 cc.) volume, the maximum pressure reduction obtainable would be to approximately one-fourth of an atmosphere. For the purposes considered in a subsequent paragraph, this degree of pressure reduction may be excessive. For these purposes, a reduction to two-thirds of an atmosphere is safe and, generally, effective.

Pressure alteration may be profitably used by the patient in the following instances:

1. It may be used in perforation of the tympanic membrane as an aid to the evacuation of discharge from the middle ear prior to the instillation of ear drops. The patient is instructed to tilt his head, the affected side down, so that the discharge will tend to gravitate over the perforation. He then produces suction, as described above, once or several times, sucking the fluid into the external canal, whence it can more easily be removed by dry wiping. The head is then tilted to the opposite side and the drops instilled. The patient then repeats the maneuver of producing suction, thereby displacing the air in the middle ear with the medication in the same manner in which a Proetz displacement is used to displace air with fluid in the paranasal sinuses. By this procedure, contact of the medication with the middle ear is made much more certain than by the usual method of dropping medication into the canal.

2. It may be used in certain cases of thickening and retraction of the tympanic membrane in which the Eustachian patency has been established but the drum still fails to return to a normal appearance and position, due to intramural thickening and early adhesive tendencies. This may occasionally be seen following the acute phases of serous or purulent otitis media. In addition to office inflations, the patient may be instructed in producing air rarefaction alternating with condensation three to five times during one maneuver, the latter being repeated two or three times a day.

Pressure alteration is contraindicated in cases in which the tympanic membrane is atrophic or old perforations have healed with a thin neomembrane formation.

SUMMARY

A simple method of utilizing pressure alteration of the external auditory canal has been described. It is recommended only as an ancillary measure in certain selected patients to whom it can safely be taught.

6317 Wilshire Blvd.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D.
SHERMAN L. SHAPIRO, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

INTRODUCTION

THE SUBJECT of occupational deafness is receiving increased attention, especially in the Scandinavian literature. It is notable that Finland, small as it is geographically, has recently formulated laws requiring preoccupational examination of the hearing of those going into noisy industries, with reasonable follow-up examinations. In our country not all the states offer compensation for anything less than total deafness. In those that offer percentage loss, no doubt the insurance companies will demand some precautionary measures, if the manufacturers do not do so of their own accord.

The discovery that the presence or absence of the recruitment phenomenon in high-tone deafness is of considerable diagnostic value in discriminating between cochlear lesions and retrocochlear lesions, which are principally acoustic or angle tumors, has led to new methods of determining the presence or absence of recruitment, including some new types of audiometers, e. g., the von Békésy, and some tests that will practically force the otologist to become an acoustic physicist.

In this country insurance companies, several large industries, and the Armour Research Foundation at the Illinois College of Technology have already embarked on an investigation of deafness due to noise, and their findings are being reported in medical literature.

HEARING AIDS

Following is a list of hearing aids accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association as of May 1, 1954.

Acousticon Models A-17, A-180, & A-185
Mfr., Dictograph Products Inc.
95-25 149th St.
Jamaica 35, N. Y.

Audiotone Models 11 & 15
Mfr., Audio Company of America
5305 N. 6th St.
Phoenix, Ariz.

Audivox Models Super 67 & 70
Mfr., Audivox, Inc.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Models 500 & 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C, & 22
Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 17

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corp.
708 W. 40th St.
Minneapolis 8

Maico Maxitone

Maico Model J

Maico Top Secret Model L
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10 & T-612
Mfr., The Microtone Corporation
Ford Parkway on the Mississippi
St. Paul 1

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C

Normatone Model D-53
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Models B-15 & B-30

Otarion Models F-1 & F-3

Otarion Model G-3

Otarion Model H-1, Custom "5"
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model Y (YM, YC, & YC-7)
(Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Distr., Radioear Corporation
306 Beverly Rd., Mt. Lebanon
Pittsburgh 16

Silvertone Models H-16, J-92, & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Co.
925 S. Homan Ave., Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, 966, 977, & 988
Mfr., Sonotone Corporation
Elmsford, N. Y.

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 300-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All aforementioned hearing aids have vacuum tubes.)
Accepted hearing aids more than five years old have been omitted from this list for brevity.

Transistor Hearing Aids Accepted

Acousticon Model A-300 — 1 transistor and 2 tubes

Acousticon Model A-310 — 1 transistor and 2 tubes

Acousticon Model A-330 — 3 transistors
Mfr., Dictograph Products Inc.
95-25 149th St.
Jamaica 35, N. Y.

Audivox Model 71 — 3 transistors
Mfr., Audivox, Inc.
123 Worcester St.
Boston 18

Beltone Concerto Model — 3 transistors
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Maico Transist-Ear Model O — 3 transistors
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic "All American" Hearing Aid — 3 transistors
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Model T1 (Red Dot)	3 transistors
Microtone Model T1 (Yellow Dot)	3 transistors
Microtone Model T 31 (Micro-Mite) Mfr., The Microtone Corporation Ford Parkway on the Mississippi St. Paul 1	1 transistor and 2 tubes
Otarion Model C-15	1 transistor and 2 tubes
Otarion Model F-22 Mfr., Otarion, Inc. 4757 N. Ravenswood Ave. Chicago 40	1 transistor and 2 tubes
Radioear Model 820 Mfr., E. A. Myers & Sons, Inc. 306 Beverly Rd. Mt. Lebanon Pittsburgh 16	3 transistors
Silvertone Model H-25 Mfr., The Dahlberg Co. Golden Valley Minneapolis 22 Distr., Sears, Roebuck & Co. 925 S. Homan Ave. Chicago 7	3 transistors
Sonotone Model 1010 Mfr., Sonotone Corporation Elmsford, N. Y.	1 transistor and 2 tubes
Telex Model 954 Mfr., Telex, Inc. Telex Park St. Paul 1	1 transistor and 2 tubes
Unex Model TR-3D Mfr., Nicholas & Clark Hawthorne, Mass.	3 transistors
Zenith Model Royal-T	3 transistors
Zenith Model Super Royal-T Mfr., Zenith Radio Corporation 5801 W. Dickens Ave. Chicago 39	3 transistors

Semiportable Hearing Aids Accepted

Ambco Hearing Amplifier (Table Model)
Mfr., A. M. Brooks Co.
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Results of the use of hearing aids were studied in 1,800 patients by P. Weersma [1]; 300 answered a questionnaire. General results were good, but strong recruitment was the most important factor in limiting the use of the aids. This difficulty may be overcome by automatic volume control.

Estimation of the loss of earnings in the case of sound-conduction disturbances and sound susceptibility was made by W. Wagemann.[2] The usual compensation in bilateral loss by hearing is too low in view of the occupational loss and psychological toll. The author offers what he considers a fair scale. A good aid may reduce the damage or obviate it altogether.

J. J. Groen, of Utrecht, and J. W. Tappin, of London,[3] undertook "a comparison of the physical characteristics of five well known hearing aids with the theoretical and apparent subjective needs of the patient."

Graphs are shown of ordinary conversation and of the frequency-amplification curve in each of five instruments. The types of persons examined were those with mainly high-tone loss, those with even loss throughout the frequency range or with low-tone loss, and those with recruitment.

The results show that attempts to compensate for high-tone losses are rarely successful. Discomfort and noises are complained of. Generally, a hearing aid with a flat characteristic for high tones, cutting off at about 4000 cycles, is preferred. In general, peaks of moderately sloping sides are more favorably received by patients.

Suggested requirements are the following: Amplification frequency should not contain sharp peaks greater than 5 db.; the slope of low frequencies up to 1000 cps should be variable from 6 to 12 db. per octave, and the part of the characteristic from 1000 to 3500 cps should be flat and taper off 20 db. less at 4000 cps.

The maximum input curve must be above speech level of ordinary conversation—65 db. above threshold at lower frequencies and 45 db. at 1000 cps.

The modern magnetic telephone is preferable to the crystal earpiece, because it tends to flatten the peaks produced in the frequency-amplification curve.

The internal noise and the cord noise must be kept below 50 db. above threshold at maximum volume. Maximum amplification should be 50 db.

The principles involved in selecting a hearing aid are discussed at length by Aram Glorig.[4] The hearing aid is an audioamplification system, modified by its necessary size more than by any other factor. First consideration: What is the output versus the input? The signal is sound, which must be converted into electrical energy, amplified 100,000,000 times, and reconverted to physical energy (sound). The frequencies 300 to 3000 cps are roughly those concerned with speech. Conduction deafness causes loss in both low and high tones; perception deafness affects the higher frequencies, progressing toward the lower and also toward greater loss. These facts determine the need for amplification. Distortion inherent in microphones and receivers makes selective control of high amplification throughout a wide frequency range difficult and accounts for variations in instrument response. A high acoustic output at too narrow a peak may be useless for speech intelligibility. Extraneous noises may be caused by rubbing of the case against the clothing or by inherent electronic characteristics.

The author quotes Hallowell Davis on the essentials in an instrument: (1) power, (2) tolerability, (3) fidelity, (4) wearability, and (5) sensitivity and amplification sufficient to hear speech at conversational level.

Conduction deafness is easily overcome by sufficient power to pass the barrier to the cochlear nerve endings. In perception deafness, discrimination is the controlling influence, and the aid merely amplifies sound. The patient hears but does not understand (discriminate). Increase in intensity does not correct this completely. He does not hear the consonants, and increasing the loudness merely causes distortion. Davis and his associates studied selected deaf persons on a special instrument equipped with filters and selective amplifiers and concluded that a flat frequency response or one with a slight decibel rise at each octave above 1000 cycles gave the best type of general response. When the modern miniature receivers and microphones perform as well as the larger types, selection will be more exact. Tolerance, dynamic range recruitment phenomenon, and the psychologic reaction of the prospective wearer, all have a definite bearing on selection of a hearing aid.

The deaf constitute a disabled group having much potential man power. The Army at Walter Reed Hospital, the Navy at Philadelphia Naval Hospital, and the Veterans Adminitsration have definite programs. (No mention is made of medical clinics associated with teaching institutions.)

The method of selecting a hearing aid at the Army Audiology Center is described. It included pure-tone, speech audiometry, and psychogenic tests in a sound-proof room. The subject is screened through 7 to 10 commercially available hearing aids; performance is evaluated at threshold, at 40 db., and at 100 to 115 db. Performance is also judged by response to spondee words for threshold and to PB (phonetically balanced) words for discrimination and in the presence of static noise.

Where such expert help is not available, Glorig recommends that a friend be the tester and that four or five aids be tried before deciding on one. It is assumed that an otologist has advised that an aid be used. Statistics are given as to the percentage of crystal and magnetic receivers compared for 1947 and 1950 and for battery voltage, showing considerably less voltage necessary in 1950 and only 1% of crystal receivers in 1950 compared with 31% in 1947. Several pages of detail in the original article are necessarily omitted here.

It is interesting to note that of the many aids tried, a very high percentage were finally selected from among four makes. The article should be read in full.

Occupational deafness continues to be an important subject for investigation in Europe. Børge Larsen,[5] of Odense, Denmark, first deals with methods of study, including animal experimentation. The factors causing traumatic deafness are duration of exposure, elapsed time between exposure to noise and hearing test, the quality of the noise, exposure to vibration of machinery, the worker's condition, and previous ear diseases. Some writers believe that middle-ear disease is a defensive factor; others believe that patients with suppuration, especially with granulations, are more susceptible (Peyser). Nerve deafness appears to cause greater susceptibility to trauma. Because the early losses are in frequencies above the speech range, the patient is not aware of them early, although he may have experienced discomfort at the first exposure, but to which he had accustomed himself subjectively. Buzzing is present in about 50% of the patients. Insomnia, nervousness, and headache are mentioned in various proportions by several authors. As to diagnosis, a healthy person under the age of 50 years without a history or evidence of aural disease and without a family history of severe constitutional disease but with a history of exposure to noise and with high-tone loss is a suspect. A c5 dip is important in the early stages, first excluding intoxications, syphilis, head trauma, otosclerosis, and

retinitis pigmentosa. Larsen then discusses prophylaxis, adaptation. He does not believe that one gets an exact picture measuring in only one frequency. He mentions the Ruedi and Furrer apparatus, which is worn externally and is properly selective and does not interfere with conversation, also the Lüscher and Zwislocki defender with selective attenuation, and others. The drawback is that the majority of workers prefer to risk the loss of hearing. Perhaps the best method is preemployment examination combined with control examinations.

HEARING TESTS

Farrior and his associates [6] use a calibrated Weber test to measure the type, degree, and duration of postoperative serous labyrinthitis following fenestration surgery. The calibrated Weber test is performed by placing the bone-conduction oscillator of the audiometer in the center of the forehead and testing with intermittent ascending stimulation for the frequencies of 500, 1000, and 2000 cps. The threshold is first determined, and then the lateralization is determined at 10-db. steps to the maximum 60-db. level. The test is recorded on the routine audiogram. Preoperative tests are made and then the postoperative tests on the 1st, 7th, 14th, and 21st postoperative days. The usual postoperative course of bone-conduction threshold can be divided into three phases, namely, a primary improvement, secondary depression, and tertiary improvement. The calibrated Weber test discloses variations of lateralization from complete lateralization to the ear operated on, called ipsilateralization, to lateralization to the ear not operated on, known as contralateralization. Lateralization may occur from desirable physiological improvement of threshold or undesirable pathological recruitment. The calibrated Weber test can differentiate the two. The writers classify the various forms of lateralization as follows: Ipsilateralization is complete lateralization to the ear operated on from threshold to maximum intensity. Bilateralization is a failure to lateralize, with the bone conduction the same in the two ears and no recruitment. This can occur when there is a failure of the threshold to improve, as in the case of round-window fixation or operative hemorrhage into the inner ear. Partial contralateralization is a partial shift of lateralization to the ear not operated on. The sound is heard first in the ear not operated on, then in both ears, and then in the ear operated on. Such a finding indicates a serous labyrinthitis, with a depression in threshold but increased sensitivity to greater volume. This pathological lateralization to the ear operated on is due to undesirable recruitment and can only be determined with the calibrated Weber test. Complete contralateralization is the lateralization to the ear not operated on from threshold to maximum intensity and indicates a profound depression of function in the operated ear.

Secondary variations in the lateralization of bone conduction are produced by the sterile postoperative inflammatory reaction called serous labyrinthitis, which can occur in various degrees from minimal to severe. Lateralization of the bone conduction to the ear operated on remains sustained throughout the postoperative course. In mild serous labyrinthitis there is enough secondary bone-conduction depression to lose the primary ipsilateralization (to the treated ear) and to show bilateralization but not enough to produce a shift to the opposite ear. Moderate serous labyrinthitis produces enough depression in the bone conduction to show a partial contralateralization, and the severe serous labyrinthitis produces a complete shift of lateralization to the ear not operated on. The loss of hearing for high tones

seems to have a direct correlation with the severity of the serous labyrinthitis. The calibrated Weber test also disclosed a tertiary improvement of bone conduction averaging 12.9 db. for the three speech frequencies, the greatest improvement being at the frequency of 2000 cps.

The writers hope that the calibrated Weber test will prove to be of academic value in the detailed study of postoperative serous labyrinthitis. They suggest that the following criteria be followed for the evaluation of postoperative serous labyrinthitis:

1. The presence or absence of primary improvement
2. The degree of secondary variations in bone conduction as manifested by shifts of lateralization, depression of threshold, diplacusis, or recruitment
3. The time and degree of tertiary improvement in bone conduction
4. The six-week improvement in air conduction for high tones and for the usual speech frequencies
5. The incidence of terminal nerve deafness

Jerger [7] introduces a new test for cochlear reserve in the selection of patients for fenestration surgery. This test incorporates both the DL (difference limen) test of Lüscher and the degree of recruitment, resulting in a test known as the DL difference test. The difference limen is measured at two levels above threshold, 10 and 40 db. The difference between these two difference limens reflects the degree of recruitment at the frequency being tested. Specifically, as the degree of recruitment increases, the magnitude of the difference decreases. The test was first standardized on nonotosclerotic perceptive losses with recruitment in order to establish an exact relationship between the amount of DL difference and sensorineural loss in decibels. Eighteen cases were tested, establishing a standard from which a table or chart was set up showing the relationship between the DL difference and the predicted sensorineural loss, or secondary nerve change, in decibels. The DL difference test was administered to 55 preoperative fenestration patients. The test was done at three frequencies, 500, 1000, and 2000 cps. The prediction of the postoperative level at each of these frequencies was made by adding 25 db., which is an assumed conductive deficit in these cases, to the sensorineural loss, as predicted in the table. These results were compared with the predicted results arrived at by using Shambaugh's test, in which the bone conduction corrected for the "Carhart notch" is used as the basis, to which 25 db. is added. In 24 of the 55 cases the DL difference test was slightly superior to the bone-conduction prediction. In the 24 cases in which the predicted result by both methods was greater than 29 db., the bone-conduction prediction was more accurate than the DL difference test by more than 5 db. in 2 cases, whereas the DL difference test was more accurate by more than 5 db. in 6 cases. This test is a valuable addition to the other two tests in predicting postoperative fenestration results. The other tests are the Shambaugh bone audiometry test and the speech discrimination, or PB score, introduced by Walsh. This test utilizes the recruitment phenomenon independent of bone and speech audiometry. Further research is necessary before the test will be simplified for routine clinical use.

Sortini and Flake [8] describe a speech audiometry test used for testing preschool children. A description of the equipment used is given. This technique was used for testing children at the Hearing Clinic of the Children's Medical Center in

Boston. The child is brought into the testing room and given a tray containing various toys, which he is allowed to handle and name. This establishes whether the child has sufficient speech for test cooperation and, secondly, his terminology for the test items used. The free-field threshold is first obtained. The child is told by the tester that he will hear the tester's voice from a big box in the room and that they will play a game. If he does not hear the voice, the tester will make his voice louder. The tester then gives instructions, such as "Give Mother the baby" or "Take the cowboy from Mother." In this way a speech-reception threshold is obtained. The earphones are then used with the same technique to obtain speech-reception thresholds for each ear individually by air conduction and then, if indicated, by bone conduction. If the child refuses to cooperate, a retest is recommended. When satisfactory results are unobtainable and the child is known to have no speech or lacks ability to cooperate, a psychogalvanic skin resistance audiometric test is done. The advantages of the test are that it requires short administration time, insures a high degree of subject cooperation, and holds the child's attention for the whole testing period. It is a reliable evaluation of the preschool child's ability to hear and understand speech from an active communication standpoint. It eliminates the need for the pure-tone audiometer test, which is more time-consuming and less reliable at this preschool age level.

Bangs and Mullins [9] write a very instructive and informative article reviewing the audiologic techniques now in use for testing for the presence of recruitment in hearing and evaluating these tests in terms of their validity and availability. The existing methods are described, including the procedure, the necessary equipment, and the advantages and disadvantages of each test. The tests discussed include both loudness balance testing and difference limen testing.

The writers made a comparison of four of these methods, namely, the range of loudness method, the Békésy test, the Lüscher-Zwislocki test, and the Denes-Naunton method. Two groups of subjects were tested, a normal and a deafened group. No subject was used who showed more than a 10-db. loss at any single frequency. Bangs and Mullins found that the most discriminating technique was the range of comfortable loudness method, with the uncomfortable loudness to threshold measure easily the best of the three measures in this method. The Békésy test seems to be the next most valid technique followed by Lüscher's general technique and then the general Denes-Naunton technique. These conclusions were arrived at by analyzing the necessary characteristics of a good recruitment test. They are as follows: 1. It must be as valid as possible, namely, it must measure recruitment. 2. It should be applicable to all types of hearing losses. 3. It should be inexpensive, the cost being within the scope of the budget of the audiologist or the otologist. 4. It should be simple to administer and to interpret. In view of these considerations, the most desirable technique for measuring recruitment is the range of comfortable loudness technique. This article should be read by those interested in the technique and evaluation of the tests employed for measuring recruitment.

Palva and his associates[10] review the subject of noise audiometry and give the methods by which the intensity of a noise can be measured and specified. It has been shown that the masking effect of the noise at very low levels is evidenced only for middle frequencies where the ear is most sensitive. As the noise level is increased above 30 db. per cycle, the equal masking contours are parallel, showing that at all frequencies a 10-db. increase in noise level corresponds to a 10-db. increase in the

masked threshold. On this information it can be stated that the relation between the masking of a pure tone by a noise and the intensity of the noise is linear above the low levels. The authors point out that theoretically, particularly with respect to the concept of the critical band, the masked thresholds for patients with different kinds of hearing loss should be the same, providing that the quiet threshold is not higher than the normal masked threshold. The experimental data reported show that the masked thresholds for pure tones ranging in frequency from 250 to 8000 cps taken in the presence of a white noise, whose overall level between 0 and 7000 cps is 100 db., all lie very close to one another, mostly within a 5-db. range and at higher frequencies within a 10-db. range. They conclude that the masked threshold is not a useful indicator for differential diagnosis.

Harris and his associates [11] describe and used a metal band helmet to which was attached a bone-conduction vibrator. The purpose of this helmet was to make it possible to adjust and standardize the position of the vibrator tip on the mastoid process of either side, the angle of thrust, and the applied force. They made repeated tests both with the helmet and the hand-held vibrator on experienced and inexperienced subjects at six octaves from 256 through 8192 cps. The difference between successive thresholds was somewhat less with the helmet than without. The helmet significantly reduced variance among thresholds repeated over extended periods. The authors recommend that the applied force be standardized between 200 and 400 gm.

Simonton and Hedgecock [12] made a study of the acuity for voice signals against a background of noise. This investigation was made to gain information for evaluating the hearing in a group of airline pilots and for making recommendations concerning their continued employment by the airline. The masking noise used was set up to approximate the over-all intensity and the frequency characteristic of noise present in the cockpit of an airplane in flight. The tests were made in a quiet sound-insulated room having an ambient noise level of 35 db. A pure-tone audiogram was made and the speech reception threshold for the Harvard spondee word lists determined. The same tests were repeated in the presence of the masking noise. Twelve young adults with normal hearing were tested as a control group. This group showed that the threshold of hearing for speech signals in quiet surroundings approximated the average thresholds for pure tones in the speech frequencies. Also the threshold for speech reception delivered against a masking noise of 98.5 db. was 60 db. higher than for the same signals delivered in the quiet room. These findings show that for normal ears the increase in threshold approximated the increase in masking noise present in the two conditions of the test. For defective ears the following conclusions were reached. The type of hearing loss and the threshold level are both required to estimate the ability to discriminate in noise. Middle-ear conduction loss of hearing does not reduce discrimination of speech in environments of noise. Loss of hearing due to impaired perception of sound showed a moderate loss of discrimination in noise. There was a wide individual variation in this group. Bilateral cochlear deafness showed a severe loss of discrimination both in quiet and in noise. However, in unilateral cochlear deafness, discrimination scores from a better ear were not affected in either the quiet or noisy environments. A favorable ratio of intensity is necessary between signal and noise for efficient hearing in noise.

Sambataro and Pestalozza[13] determined the effect of white noise in connection with speech tests on six subjects with otosclerosis after exposure to white noise at 60 db. above threshold intensity level for 15 minutes. In work done on normal hearing persons under similar conditions, reported last year by Sambataro and Pestalozza (Masking and Fatigue Effect on White Noise in Connection with Speech Tests, *Laryngoscope* **62:** 1197 [Nov.] 1952), the threshold shift was small for low levels by white noise becoming larger as the white noise was increased in volume. The normal-hearing subject after exposure to the white noise at a high intensity level shows an impairment of 5 db. for logotomes along the entire length of the articulation curve. The threshold shift in cases of otosclerosis is lower for low intensities of white noise and similar to normal for medium and high intensities. The improvement in threshold always encountered in normal-hearing subjects is not encountered in otosclerosis. This gives value to the hypothesis that the fatigue of the stapedius muscle is a cause of this phenomenon in the normal-hearing ear.

The subject of nonorganic deafness, monaural or biaural, is discussed by Heller and Lindenberg.[14] This symptom is observed not infrequently in clinical experience. The authors review the various tests which can be utilized to diagnose this condition, such as repeating the pure-tone audiometric tests, the Doerfler and Stewart test, and the Lombard and Stenger tests, the latter done with tuning forks or special audiometric equipment. The study of the entire patient, his history, behavior, speech and physical status will be a great aid in making an accurate diagnosis. The psychogalvanic objective test is a good test and frequently clinically reliable. The hearing tests will not expose the motivation for the complaint of nonorganic deafness. This must be uncovered by psychiatric investigation.

Curry[15] used the ear-choice technique for obtaining threshold responses in 20 subjects ranging from 13 to 18 years of age who had known serious hearing losses. This technique had been used previously on normal subjects. The technique is as follows: Headphones are placed on the subject's head, and both techniques, namely, the standard and ear-choice, are used to get the discrete frequency threshold audiogram without moving the phones. The subject is asked to respond when he hears the stimulus by touching the earphone which carries the sound. The frequencies were tested in the following order: 1024, 2048, 4096, 8192, 512, and 256 cps. To find the threshold, the initial stimulus is set at a level of 30 db. and presented first to one ear and then to the other. This is started at 1024 cycles. If the sound is heard in both ears, then the dial is shifted to 2048, leaving the attenuator dial at 30 db. sensation level. It is then reduced to 20 db. for a complete sweep and then to 10 db. and lower intensities. If there is a failure of response at 30 db., the sensation level is increased at 5-db. steps until a consistent response is obtained. Then the attenuator is reset to the original sensation level at 30 db. and the sweep continued. When a threshold is obtained, it is rechecked at the next lower sweep and if found correct is eliminated from further testing. If the initial stimulus of 30 db. is inaudible in either or both ears, the new threshold is found immediately and then the stimulus level returned to the initial sensation level, 30 db., for further testing of the other frequencies. Curry finds that this test may be completed in an extremely short time and that the motivation of young children seems to be strong in this technique. This technique has been shown to be as accurate a measurement as the standard pure-tone audiometry test in normal-hearing adults and in adolescents

with known hearing losses. This test adds another check to the response of the patient, as it inquires as to which ear he hears the sound in. The ear-choice technique is very simple to administer.

Weille and Gargano[16] present some of the present-day concepts of bone conduction held by the acoustic psychologists, which should be seriously evaluated by the otologist for clinical use. Four cases were studied, each having one "dead ear" functioning. Their findings support Békésy's theories concerning bone conduction. Inertia bone conduction is produced by movement of the skull relative to the footplate of the stapes as a result of inertia of the ossicular chain. It postulates simultaneous stimulation of both ears because of skull movement roughly toward one set of ossicles and away from the other, the ossicular lag being due to inertia affecting one ossicular chain about the same as the other. This hypothesis is substantiated in testing cases with one totally destroyed ear and the other normal; the hearing ear is stimulated equally easily from either mastoid region. Although inertia bone conduction is believed to be most important at low frequencies and compression conduction at high frequencies, both are theoretically present throughout most of the audiometric scale. Therefore no bone-conduction audiogram is valid if both ears are functioning unless masking is done, because of uncertainty as to which ear is responding to the test tone. Quantitative masking will prevent overmasking and will give the examiner the information as to the approximate alteration in the threshold of the masked ear. The saw-tooth noise built into some audiometers is acceptable for practical purposes. It has its zero setting at the "just audible level." The white-noise masking device (100 to 1000 cps) with a wide and nearly flat amplification curve is the masking noise of choice. This has its zero setting about 15 to 20 db. lower than saw-tooth noise. A white-noise sound pressure must reach approximately 30 db. in either ear before any threshold is affected; above this level the threshold is altered decibel for decibel. Thirty decibels is required for sound to go around the head to the opposite ear, if it is uncovered. By adding this 30 db. to the 30 db. sound pressure required, as mentioned before, a minimal masking sound of about 60 db. on the ear not being tested is allowable in all bone-conduction scores, if both ears are "normal." If the ear undergoing the threshold test is deaf to air-conducted sound, the amount of this deafness in decibels may be added to the masking sound if the examiner so desires. The air-conduction receiver begins to generate bone-conducted sound at about 60 db. If the 30 db. allowable noise not affecting threshold is added to the 60 db., it is concluded that shadow bone-conduction masking of the ear under threshold testing can be excluded. Békésy's fundamental concepts apply equally to tuning forks used as bone oscillators. The presence or absence of compression bone conduction probably explains the presence or absence of a "drop through the skull" in sound pressure in bone-conduction audiograms. The variability of results in bone-conduction audiometric tests retested is controversial as to magnitude. The best scores of multiple determinations over a period of time are the probable index of true function. A means of checking the validity of bone-conduction measurements may be repeated phonetically balanced speech audiograms carried out at a high sound pressure level, usually about 100 db. There are no widely accepted standards for calibration of the bone conduction zero reference level built into audiometers. Therefore substantial variations may occur from one make to another. Since there

are no basic acceptable standards agreed upon by the Bureau of Standards, some commercial bone-conduction oscillators may not be the instruments of precision that they are reported to be.

In a nine-page article LePage [17] gives an excellent summary of pure-tone audiometry as it is practiced in the otologic service of Professor Destree at Liége, Belgium. The paper cannot well be summarized but should be read in the original as a concise description of the methods being used in an up-to-date clinic on the continent. Of interest is the modification of the techniques and ideas in this field, which at first followed closely those in this country. Both air and bone conduction are tested for each frequency, the latter including not only relative but also absolute bone conduction. The writer takes pains to explain that the zero line for this is adjusted to relative bone conduction on American audiometers, while in the Peters (English) audiometer, which he uses, zero is adjusted to absolute bone conduction. The results of both methods are combined for the frequencies 256, 512, and 1024 to obtain the index 0 of Sullivan, which normally amounts to 35 to 45 db.; less at 20 db. is indicative of stapes ankylosis. For masking where the difference is less than 40 db., simple occlusion of the other ear with the finger is employed for air audiograms; where the difference is greater than 40 db., a pneumatic device is used exerting a pressure of 15 gm. against the drum membrane. The device is not used in stapes ankylosis or aural suppuration. For bone-conduction tests the customary white noise is employed on the other ear. The audiometric Rinne, Schwabach, Weber, and Gelle are described. For the latter a technique involving a sphygmomanometer is used, and only the 512 frequency is tested; 5 to 15 mm. of mercury is sufficient in a normal person to cause a disappearance of the sound, and persistence of the latter when a pressure of over 20 mm. is used is a sign of stapes ankylosis. A description is given, also, of the method of Boisnier, in which bone conduction over the mastoid process is compared with that over the seventh cervical vertebra, the sternum, and other locations; under certain circumstances this test will furnish a diagnosis of otosclerosis. Recruitment and the difference limen test of Lüscher are explained in detail; they are routinely tested on the Peters audiometer. Tabular summaries and good graphic illustrations accompany the article.

Croatto and Croatto-Martinoli [18] give a summary of group testing methods, which are stated to be the first group hearing tests conducted in Italy, as well as their personal experience in this field. Their material consisted of 1,125 children in the first five classes in three elementary schools of Padua. They used pure tones according to the technique of the Massachusetts hearing test as well as tests with a sweep frequency audiometer. The first method was made in two sessions, using a Maico D-9 with 40 receivers. At the first session three tones, 512, 1024, and 4096, were tested; at the second one the 11000 tone was added. Preliminary detailed instructions were given to the children, who marked cards in front of them with an "x" indicating whether they heard the frequency and intensity or not. Those children who showed more than two mistakes were rechecked by the sweep frequency method, starting 10 db. above threshold. All children who showed a loss of 20 db. in two frequencies or 30 db. in one frequency were sent for a complete audiometric study in a sound-proof room; those whose final study indicated a serious hearing deficiency were turned over for guidance to the social service department. The authors intend to continue their studies to cover the remaining schools of Padua.

Pirodda and Celletti [19] discuss the differential diagnosis of various conditions which produce perception deafness. They analyze 10 cases of this type of hearing loss including lesions of the end-organ as well as the cochlear nerve and central auditory pathways. Their conclusion confirms the work of Dix and Hallpike and Hood, namely, that the recruitment phenomenon is strictly an attribute of the organ of Corti and is absent in more central lesions. A review of the literature and audiograms, as well as detailed case histories of the authors' own patients, is included.

An attempt to follow the outcome as to hearing ability following radical mastoidectomy is made by Di Nicola and Pinto.[20] The authors call attention to the variations in the state of the hearing which are produced by this procedure. Statistics are quoted from Bjorkman, White, Jensen, Nielsen, Alfoedy, and others—statistics which vary considerably with different authors. They stress recent figures from a series of cases by Pietrantoni involving patients operated upon either in the classic manner or by means of a conservative radical, also with and without skin grafts. His best results were on patients who had a classic radical with skin grafting; in 57.1% the hearing was improved, in 31.4% it was made worse, and in 11% it was unchanged.

The authors studied a series of 14 patients operated upon by the method of Wolf in order to follow the course of the hearing function during a four-month period after the operation. Audiograms were made beginning with the eighth day and continued at regular and frequent intervals. They found that the hearing function in these patients showed certain definite characteristics for all frequencies. During the first two weeks there was a rapid decline; at the end of this period this was arrested, and a slow recovery took place. In some cases this did not stop until the hearing exceeded that which the patient had before the operation; in others it equaled the preoperative level, and in some the hearing never recovered to the preoperative extent. Further observations for a year will be continued.

Van Eyck [21] discusses auditory fatigue. As Hood, he believes that a distinction must be made between fatigue proper, which results from moderate intensities, even as low as 20 db., and exposure to sounds of high intensity, which produce, especially if repeated, actual pathologic changes in the hearing end-organ. There exists for each frequency a "critical intensity" which is not necessarily synonymous with the pain threshold. Sounds below this level produce fatigue which is in accordance with auditory nervous physiology and from which complete recovery takes place, while sounds above this level produce a hearing loss which is either irreversible or only partially reversible; it is this type of stimulus which is responsible for occupational deafness.

Van Eyck also makes a distinction between poststimulation fatigue and prestimulation fatigue occurring during the auditory stimulus; the latter reaches its maximum hearing loss after a three-minute exposure to the sound and is directly proportional to the intensity of this sound. The phenomenon is not related to fatigue of the musculature of the middle ear, as it occurs in pigeons which have been subjected to curare; it is probably an adaptation phenomenon on the part of the fibers of the auditory nerve. It must be borne in mind during hearing tests that fatigue phenomena play a role, and, in particular, this fact must be considered in interpreting the results of recruitment tests.

Aubry [22] reports on a method of testing the hearing by means of a watch which is placed on the microphone of the audiometer. Thresholds must be determined for

each watch; the author's instrument was found to be 15 db. by air and 20 db. by bone and its frequency range to be that of vowels with a maximum range of 3000 d. v. Tables are given comparing results of this method, which the author claims to be simple yet remarkably precise, with pure-tone, whispered-voice, and vocal audiometry.

Veckmans [23] contributes a monograph on audiometry. Of the 282 pages comprising this work, two-thirds deal with theoretical considerations involving sound, the physiologic mechanism, theories for its reception, and similar considerations. The remainder discusses techniques, diagnosis, speech audiometry, hearing aids, and compensation for hearing disability.

Some points of interest involving the more practical portions of the monograph may be mentioned. The author feels that the receiver, even with the rubber pad, does not fulfill strictly the scientific requirements for air conduction but admits its convenience; some space is devoted to a description of a method whereby a loud speaker can be used for this purpose and the opposite ear be excluded. Recruitment is discussed in detail and all the methods described; the author favors that of Lüscher. There is little enthusiasm for masking, and the flat statement is made that insofar as testing bone conduction is concerned, this is a fallacy, as masking gives a false threshold when applied to the better ear, even as a distorted picture is obtained without masking. The author is highly in favor of the method of van Dishoek (continuous frequency variation while keeping an even intensity) as an additional check. Speech is tested by means of material from phonograph records transmitted by loud speaker and received binaurally. Veckmans feels that both masking and telephone receivers distort the results of speech tests.

Miodonski,[24] as a result of previous experiments with audiometric curves made by testing through the paranasal sinuses and comparing these with typical audiograms in cases of stapes fixation, arrived at a very simple test for differentiating between conductive and perceptive deafness. This is done by having the examiner occlude both external meatuses with the fingers and asking the patient to repeat conversational speech. Those with conductive deafness will be able to do about as well as before the occlusion; those with perceptive deafness will not be able to do it at all. The test is offered as a rough test for outpatients and for the use of the nonspecialist.

A method of diagnosing recruitment by means of aural harmonics is offered by Sokolowski.[25] After discussing the methods of Fowler and others, he recalls that a pure tone of satisfactory intensity registers not only the fundamental tone but also a series of harmonics or overtones which in terms of frequency are multiples of the frequency of the original tone. In practice he used a frequency of 500 kilocycles sent by air conduction and an auxiliary tone very near the frequency of 1,000 kilocycles, for example, 1,003 kilocycles (the method requires a continuous frequency audiometer) sent by the bone-conduction receiver through the mastoid process; in this way distinct beats (in this case three) were heard from, which were defined by the patient as interruptions of the tone. The author found that in normal subjects beats appeared at an intensity of 45 to 50 db. in the fundamental tone and 40 db. in the auxiliary tone. The beats disappeared below this intensity. In conduction deafness the beats appeared when the above figure for the normal was added to the threshold of the patient. In perception deafness the beats appeared in

the deaf ear at the intensity of a fundamental tone only slightly greater than the intensity of the threshold for 500 kilocycles, if the loss was greater than 45 db. Audiograms and comparisons with Fowler's test are attached.

Faure, M. Portmann, and C. Portmann [26] continue certain studies on the psychogalvanic reflex in audiometry, which were described previously by two of the authors. Their conclusion is that with a meticulous technique an audiogram obtained by this means is an exact representation of the hearing loss caused by a peripheral lesion. In central deafness the results require a more delicate interpretation but may still be of value when they are considered within the context of electroencephalography. The authors make the point that the procedure is valuable in children, malingerers, and other cases, inasmuch as the great majority of cases are on a peripheral basis.

Greiner, Rohmer, and Weill[27] discuss the clinical application of the Lüscher difference limen test. They call attention to the fact that either variations in the perception of intensity differences or in rhythm may be present. The abnormalities in the perception of rhythm offer considerable possibility in the discovery of central auditory lesions. They summarize the few existing observations and add a case of their own with illustrative audiograms.

Azzi [28] calls attention to the fact that a person with normal hearing can distinguish the voice of someone who is speaking to him, even against a noisy background including other people who are conversing. This phenomenon is, of course, limited by the intensity of the masking noise. It has also been observed that persons with hearing diminution caused by lesions of the perceptive organs have considerable difficulty under these circumstances, while those with conduction impairment are not bothered unless the level of the masking voice approaches that of the person whom they are trying to hear. This phenomenon gave Azzi the idea of testing the power of the ear to distinguish a given voice from a background of another voice as a guide for determining the type of hearing impairment. On a tape recorder there is registered in an even tone a "first voice" which pronounces two-digit numbers at four-second intervals; a little later as this person begins to speak, a second voice is interposed pronouncing other numbers with no intervals between them. This "second voice" is at first in a low intensity, but gradually this is increased until the masking voice is as strong as the "first voice." The record is connected with the audiometer, and the speech is presented at a level 20 db. above the patient's threshold. Both male and female voices were used as "first" and "masking" voices and a normal level of masking voice intensity established. Specific instructions were given to each patient to pay attention only to the original voice and to record the numbers on a given form. Through a check of the results with the pure-tone audiograms, it was found that the original premise was correct, i. e., persons with perception deafness were considerably hindered by the masking voices, while those with conduction deafness could distinguish the "first voice" perfectly from the second, or "masking," voice.

Moretto [29] discusses the various methods available for testing the hearing capacity of young children. After mentioning Urbantschich's harmonica, conditioning methods (such as Dix and Hallpike's "peep-show"), and the simple instruments used by Utley, he proposes a simple means of testing the hearing by means of speech audiometry modified from the suggestions of Keaster and Westlake. This is as follows: A folder of four pages is made; the first page contains instructions to the

parents, and the other three pages contain 12 drawings of simple objects with which a young child would be acquainted. The instructions to the parents are, in effect, to teach the child to put a finger on the object when the name of the object is pronounced. This is performed over a period of several days, first standing in front of the object, then at various distances behind the patient. When the parental instruction to the child has been completed, the latter is brought to the clinic and the examination conducted in a soundproof room which is connected to a high-fidelity loud speaker, with use of the same commands but of graduated intensity. If the patient has poor hearing, this can be done directly in front of him in order to ascertain the capacity for learning lipreading, and it is also possible to test monaural hearing by means of receivers.

The technique of speech audiometry and a simple speech audiometer with masking generator for clinical use are described by Knight and Littler.[30] The technique in conduction and perceptive deafness and for the selection of a hearing aid are described, as is a simple speech audiometer with masking generator and its calibration. Instructions for technicians are included, as are word articulation test records.

The value of speech audiometry in diagnosis and prognosis is emphasized by Walsh.[31] Speech is used instead of pure tones. The hearing is measured at threshold and at suprathreshold intensities. The examiner knows whether the sound is heard without relying on the patient's signal. The recruitment phenomenon is present in cochlear lesions, e. g., Ménière's disease, and absent in lesions medial to the cochlea. In end-organ lesions speech inteligibility reaches a maximum and then declines with increases of intensity. In nerve fiber degeneration speech becomes more and more intelligible with increased intensity of sound. An appropriate apparatus costs about $400.

Speech audiometry measures ability to hear speech not only at threshold but also at suprathreshold intensities of everyday life. It is more satisfactory in predicting results of fenestration and determining when a hearing aid will be of value. It is an added test in the differential diagnosis of Ménière's disease and eighth nerve lesions proximal to the cochlea.

Improvement of hearing in the unoperated ear after fenestration is reported by H. E. Zangemeister.[32] He believes that apparently improved hearing in the ear not operated on is really due to improved bone conduction in the ear operated on.

Using an Amplivox audiometer for estimating the threshold of hearing for pure tones and the speech reproducing system used by the Electro-Acoustic Committee of the Medical Research Council (recorded speech was used) for speech testing, Terence Cawthorne and R. M. Harvey [33] tested 150 cases of conductive deafness, 180 of perceptive deafness (3 of which were due to streptomycin intoxication, 40 to Ménière's disease, 12 to trauma, and 88 to unknown causes and classified as presbyacusis), 31 of mixed deafness, and 70 cases after fenestration. The results were compared for hearing of pure tones and for speech.

In pure conduction deafness there was close correlation between hearing for pure tones and for speech. In cases of mixed deafness the relationship was not so close. In perceptive deafness the ability to understand speech often bears little relationship to hearing for pure tones, especially in Ménière's disease (end-organ deafness). This is also true of traumatic deafness. In otosclerosis the speech curve lies parallel to normal after fenestration. The speech curve usually retains this relationship especially well in favorable cases.

The authors are investigating hearing for pure tones in a free field in cases of otosclerosis, the results of which they will publish soon.

REHABILITATION

Hoople and DiCarlo [34] present some of the basic and essential requirements for the formation of a hearing and speech center. The formation of such a center depends primarily on the willingness of the otologist to work and take a vital interest. The secondary requirements, as the housing, equipment, and personnel, will follow. Next to the otologist and medical staff in importance is the audiologist. The various types of equipment necessary and a simple construction plan for a sound-treated booth are described. The cost of administering such a program will be negligible in terms of a total budget, but in terms of salvaging human self-esteem, alleviating human suffering, and providing for physical, intellectual, emotional, and social-civic growth, there can be no greater remuneration.

Newton [36] reports on how a hearing conservation program was started and developed in Arizona. Some of the unique differences from other conservation programs are discussed, such as the sponsorship by two private societies, the State Medical Association and the Arizona Society for Crippled Children. They had an intensive state-wide training program for audiometrists, parents, and teachers on "Hearing Loss in Children." They also had a strong state-wide continuous public education program. Volunteers were used for testing.

Kincaide [36] describes the part that he and his fellow otologists and the members of the State Medical Association played in the development of the hearing conservation program in Arizona. The educational follow-up program is described by Cruthirds.[37]

Sataloff [38] feels that the management of the patient with nerve deafness is primarily the responsibility of the otologist and that most aural rehabilitation should be conducted in the private office of every otologist. He should give the patient a clear understanding of his organic pathology, psychologically adjust him, fit the hearing aid where indicated, and give auditory training to teach the patient how he can most effectively use his residual hearing with and without a hearing aid. Speechreading, when necessary, is not within the scope of the otologist. The fitting of the hearing aid comprises making the impression for the ear mold and either testing with several standard hearing aids or having the patient visit several recognized hearing-aid companies, trying each one out at home. The patient brings one or two that he has found most satisfactory, and the otologist then tests the aid with the patient, using the various spondee words and PB lists. If the otologist is assured that amplification and discrimination are satisfactory, he advises the patient to purchase the aid. Once it is purchased, he should advise the patient on auditory training at home. Simple methods are used, such as having someone read to the patient every day as he uses the aid without watching the reader's lips. He should also be instructed on how to use the aid on the telephone if necessary. Psychological adjustment is also necessary for both the patient and his family and friends. The writer feels that the future of otology to a great degree will be influenced by the ability of otologists individually to solve the problems of the patient with nerve deafness.

Fowler [39] tells the story of the birth and growth of the work of organizations for the hard of hearing. He especially stresses the work of the American Hearing Society and deplores the fact that so few otologists are members of the society. All

organizations for the hard of hearing need their guidance. There are several problems which require special consideration. One of these is the question of heredity of deafness and factors influencing the activation and exacerbation of the lesions of otosclerosis. Another problem is to overcome unwarranted discrimination against the hard of hearing in industry and in certain professions. Medicolegal disability associated with hearing losses incurred in accidents or from exposure to industrial noises is another acute problem. Speech audiometry is now being favorably considered for assessing this disability. The time appears to be ripe for standardization of speech audiometry for this purpose in correlation with other diagnostic and audiometric procedures. The hearing capacity of an individual must be determined by clinical observation and audiometric observations checked with tests for speech hearing at different levels. No arbitrary procedures, as age or averages, can take their place. The past 35 years has shown an increasing increment of understanding of the problems of deafness, due to contributions of many and the growth and work of organizations for the hard of hearing. Fowler makes an appeal to the otologists, audiologists, the hearing-aid manufacturers, salesmen, and everyone to cooperate with and help the American Hearing Society, which has done so much for the hard of hearing everywhere.

Kobrak [40] discusses the treatment of deafness by prosthesis. He describes three types of prostheses—the ear drum type, which covers the perforated ear drum; the tympanal type, which is placed in the niche of the round window or oval window or against a surgical fenestra, and the ossicular prosthesis, which displaces the destroyed ear drum and ossicular chain. Kobrak describes the use of an oxycholesterol-petrolatum (Aquaphor) ointment as a satisfactory tympanal prosthesis. It fits the requirements for a good prosthesis in that it can carry moisture and hold it in the proper position and shape. It is nonirritating and can be reactivated by instilling a drop of fluid, such as an isotonic sodium chloride solution, into the middle-ear cavity. It is especially of great value in cases in which the niche of the round window is accessible. The ointment should be introduced under aseptic precautions and can be left in the ear indefinitely. The ossicular prosthesis (diaphragm-rod prosthesis) provides good hearing in some cases. The technical difficulties in making, fitting, and retaining the diaphragm-rod in place have kept it from receiving general acceptance. A mechanical prosthesis should be tried in every suitable patient.

Ronkin [41] reports the results of 68 cases of low-tone deafness and tinnitus in which mandibular repositioning was indicated and carried out. The otologic investigation was carried out by the staff at the New York Eye and Ear Infirmary. Ten cases showed an improvement of 10 db. or more; 12 cases of 5 to 10 db. improvement in hearing, and 9 cases of 0 to 5 db. improvement. In no case was there an increase in hearing loss. Several cases showed spectacular improvement both in hearing and in the loss of their tinnitus.

Kinney [42] gives a report of the hearing conditions of 3,622 children examined during the past 15 years in the otological diagnostic clinic at the Alexander Graham Bell School in Cleveland. The ages of the children varied from 2½ to 17 years, with most examinations made between the ages of 8 and 11. Six hundred twenty-seven cases had normal hearing, and 367 had no usable hearing. This left a group of 2,628 children whose hearing loss was analyzed. The ratio of conduction loss cases to perception loss was 2 to 1. A large percentage of the conduction loss cases were due to otitis media which could have been prevented. Middle-ear effusion as a cause

for hearing loss in children is on the increase. Measles virus as a cause for perception loss in children is probably of greater importance than has been previously reported. Progressive perception hearing loss in children, although not frequently encountered, is of serious consequence when it occurs and may be the result of some of our modern therapeutic endeavors.

D. Myers [43] pleads for careful consideration of the etiology and diagnosis of deafness in children. Deafness may be considered as a nonfunctional sense of hearing for ordinary purposes of life, congenital or adventitious. Etiology is important from the standpoint of prevention. The early diagnosis of deafness may be inferred by limited speech development. More definite tests, especially the galvanic skin test, are discussed.

Factors of differential diagnosis between deafness and infantile aphasia, autism, and feeblemindedness are discussed.

Finland is moving forward in the prevention of occupational deafness. Urpo Siirala [44] describes the draft of a new noise-control act to take effect January, 1953. Scientific studies have shown that loud noise causes deafness, nervousness, and fatigue, lowers the efficiency of workers, and lessens attentiveness, resulting in increased accidents. Ninety phons is considered the limit of risk. The law does not apply to manufacturers self-employed or to those employing only a few designated relatives.

Noisy operations must be replaced by less noisy ones and, where practicable, rest periods provided. Noisy machinery must be insulated from the building, walls and partitions provided as necessary, and antinoise material and structure provided.

Where conditions do not permit sufficient reduction of noise, ear protectors shall be used. Medical examinations, preferably by an otologist, are required at the beginning of employment and yearly thereafter.

Sections 9, 10, 11, and 12 describe the methods of enforcement of the law and penalties for violations.

Alfred Peyser,[45] of Stockholm, offers an audiometric fatigue test for preoccupational otologic examination of persons seeking employment in noisy industry. It consists of an audiometric test by air at 1024 cps at 100 db. for three minutes, a 15-second pause, and a retest at 1024 cps to measure the loss; duration of tinnitus is less than or more than 15 seconds. After an interval of 30 to 60 minutes, a repetition of the above method is carried out by bone conduction. Peyser then classifies the applicant as follows: loss of 5 db. or less, resistant; loss of 5 to 10 db. by bone, suspect; loss of more than 10 db. by air and 5 db. by bone, oversensitive, and evidence of irritation of long duration, oversensitive.

The author also suggests that these tests plus the usual otological examination be given to all young candidates entering trade schools.

He describes how he arrived at the above figures by various experiences and concludes that to prevent irreversible loss of hearing in more than 75% of workers, preemployment examinations by otologists should be organized. The audiogram alone is insufficient, and auditory fatigue tests are necessary to eliminate persons sensitive to noise.

In chronic suppuration, mucous membrane thickening and granulations make the patient peculiarly susceptible to noise. Peyser suggests the construction of noise-

and-vibration rooms in which standard working noise, measurable as to frequency and amplitude, can be produced. He recommends international collaboration of industrial otologists, following uniform methods.

A report on group testing of school children by pure-tone audiometry by S. F. Nielsen,[46] Chief Otologist, Esbjerg, Denmark, appears in the *Acta oto-laryngologica*. He gives a historical review of the methods of others and puts forth his own method of doing group testing by using, instead of words or numbers, a dot-and-dash method, which is easily followed by children of preschool and early school years. There is also a follow-up on those children found to have defective hearing. The entire subject is covered in detail, and the report is recommended for reading by those interested.

NOISE IN INDUSTRY AND TRAUMATIC DEAFNESS

Fox [47] reviews the problem of industrial noise and its effect on hearing as observed in industries. He also discusses the factors responsible for the hearing loss, namely, intensity or loudness of the noise, frequency spectrum of the noise, period of exposure, individual susceptibility to acoustic trauma, age of the subject, coexisting ear disease, character of the surroundings in which the noise is produced, distance from the source, and position of each ear with respect to sound waves. Before an otologist makes a diagnosis of occupational hearing loss, he should satisfy himself that the worker has an impairment characteristic of acoustic trauma, that the sound-level measurements of the working area are of intensities great enough to cause a hearing loss, and that there has been sufficient exposure time. Many methods have been used to estimate the percentage of hearing loss in these cases. The Subcommittee on Noise in Industry of the American Academy of Ophthalmology and Otolaryngology in October, 1952, announced that hereafter calibrated speech audiometry should be used for evaluating hearing loss in medicolegal cases. Fox feels that technical problems relating to equipment and procedures, as well as human factors, will be encountered which will operate at first to render speech tests difficult to use, accept, and standardize. Fox recommends that several procedures be followed in the investigation and control of industrial noise. Statistical data should be gathered, including surveys of noisy areas in industry plus preplacement audiometric and otological examinations of workers. Follow-up audiometric studies should be performed as indicated. Quantitative standards for evaluating the problems of industrial noise should be developed and set up. Proper preemployment selectivity tests to discover noise-susceptible applicants should be investigated and established, and, finally, there should be developed methods and treatment which make possible the elimination or reduction of noise entering the ears. This can be accomplished by proper planning and redesigning of machinery and tools, substitution of processes in industry for noisy operations, rotation of working personnel, and use of ear plugs or defenders. The problem of occupational hearing loss is not a partisan one. The cooperation of all involved parties is necessary for a successful solution of this problem.

Goldner [48] presents a critical evaluation of deafness in 600 shipyard workers. Four hundred three typical cases were studied carefully and their hearing values averaged. The length of employment, except for prolonged periods, did not result in a direct proportionate greater loss with greater length of employment. Older workers were more susceptible to acoustic trauma. Those workers who were more intimately connected with the noise and who worked in confined spaces showed greater hearing losses. Deafness and tinnitus were the commonest subjective com-

plaints, with tinnitus often more disturbing than the deafness. A normal response was rather common for conversation, whereas hearing for the whisper was absent in over half of the workers tested and markedly diminished in the rest. The audiogram showed a predominant loss of the higher tones.

Occupational deafness may coexist with conductive deafness, traumatic deafness due to head injury, or other types of nerve deafness. Workers with otitis media or perforations of the tympanic membranes are more susceptible to acoustic trauma. The basic criteria for the diagnosis of uncomplicated occupational deafness are the following: 1. There is a history of exposure to excessive noise. 2. The hearing impairment is bilateral and fairly symmetrical. 3. The tympanic membranes usually present a normal appearance. 4. The bone conduction is diminished in relationship to air, and the Rinne test is positive. 5. Bone conduction is diminished as compared to the normal. 6. The audiometric curve follows the pattern of the so-called "boilermaker deafness," with the greatest loss at 4096 and 8192 frequencies. 7. The Eustachian tubes are usually patent and the vestibular responses normal. The problems of prevention include the awareness of the hazard, preemployment examination and audiograms, improved industrial design and soundproofing, medical control of exposed workers, exclusion of workers more susceptible to hearing loss, and the use of efficient hearing plugs.

Sataloff [49] studied the effect of continuous noise of measured duration and frequency characteristics upon hearing acuity after prolonged exposure. This was carried out on 272 subjects who were daily exposed to a specific type of jet-engine noise. The noise and duration of exposure were correlated with any significant changes in hearing. The intensity fluctuation was between 90 and 120 db. at a frequency range of below 600 cycles. A change in threshold for any frequency was considered significant when there was a persistent change of at least 10 db., except that for a normal audiogram in which the threshold is 0 or 5 db. a significant change had to be at least 15 db. Only 154 subjects are included in this preliminary report. These conformed to the most rigid criteria possible, and those on whom complete data were not available, or who had some conductive hearing loss, or used ear protectors were excluded. Ninety-five, or 62%, had significant losses of 10 db. or more in the higher frequencies when the initial hearing survey was made. Forty-six of these showed further significant audiometric changes. Twenty-four of these were exposed to jet noise for three or more years. None showed an audiometric change at 1024 and 2048 cps. None of the subjects sustained changes in hearing acuity greater than 30 db. at 4096 and 8192 cps. This study reveals that personnel exposed to a steady-state moderately intense low-frequency noise may sustain some degree of auditory fatigue daily for many years without sustaining any significant degree of permanent hearing impairment. Subjective hearing loss and tinnitus were rarely encountered and were not related to the occupational noise being investigated. None of the changes in hearing found in any of the subjects in this study could definitely be attributed to exposure to jet-engine noise.

The observations made in this study to date show that the human ear is more resistant to injury and noise than it is usually thought to be.

Fox [50] discusses the evaluation of hearing loss in drop forge workers. Sixty-four of these workers had filed claims for alleged loss of hearing resulting from their work. Two cases were not included, as one had psychogenic deafness and the other was a malingerer.

The diagnosis was based upon three factors: the record of employment in areas of intense noise levels, the medical history, and the objective findings. There were no records of preemployment hearing or ear studies. It was necessary to rely on the applicants for this part of the history. The complaints of hearing impairment varied considerably from impairment for only high tones, such as telephone bells, to marked difficulty in hearing any speech. Clinical examination failed to reveal any local pathology. The early cases showed only a slight to moderate dip at 4,000 cps. Moderate and severe losses revealed involvement of the speech zone range of various degrees, with the greater losses in the higher frequencies. The hearing impairment affected the two ears to the same extent. Total loss of hearing was not found in any of the cases.

Two problems arose in the medicolegal evaluation of hearing loss. One was the absence of records indicating the hearing status of the worker at the time of employment, and the other was the difficulty to interpret the audiometric pure-tone losses in terms of percentage disability for capacity to hear speech. The percentage of hearing loss was determined from the pure-tone audiometric studies calculated on the American Medical Association hearing disability table. Fox believes that it would be advantageous to eliminate the 4,000-cycle frequency from the chart and assign its percentage value to the frequencies between 500 and 3,000 cps. He also states that losses of less than 20 db. should not have compensatory value, since they constitute very little handicap in hearing ordinary conversation. Deductions to compensate for the age factor were based on the World's Fair, San Diego County Fair, and United States Public Health surveys. These surveys indicate that the loss up to the age of 50 is not too significant; in the 50- to 60-year group the average loss attributed to presbycusis amounts to 5.6%, based on the American Medical Association hearing-loss chart. After age 60 the loss is somewhat higher. The hearing loss was considered permanent if present six months after complete removal of the worker from the area of loud noise. This is the opinion of the Subcommittee on Noise in Industry.

The medicolegal problems confronting the otologist indicate that authorized scientific bodies now investigating the various aspects of industrial hearing loss will have to revise and modify the presently used formulas and methods of evaluating functional hearing.

MISCELLANEOUS

Mueller [51] describes an office procedure for the fitting of hearing aids. The usual complete examination is made, including pure-tone audiometry and tuning fork tests. The live voice, both whispered and conversational, is used. One of the most useful instruments for testing voice perception qualitatively and quantitatively is the old-fashioned speaking tube. It offers a means for determining at once how well a patient might be expected to do with a modern hearing aid. If he can understand a faint whisper, almost any aid will be efficient, but if he cannot understand a fairly loud voice, no aid will give him good speech intelligibility. Whole sentences should be used. The material used should take into consideration the patient's age and education. The receiver of the hearing aid is then applied after the other tests are completed. A straight plastic nipple with a piece of pure gum tubing to assure a tight fit is placed in the ear. The patient is told to adjust the volume while the examiner keeps up a running conversation at an easy conversational level. At first several aids were tried. Now the writer uses one. He suggests that one good acceptable aid be used

with which the examiner is well acquainted and which has good service and cooperative agents. This aid can be recommended to the patient, and both the otologist and the agent should continue to keep in touch with the patient.

The actual purpose of this procedure is to demonstrate to the patient what he can expect from a hearing aid and to prepare him for his coming adventure in hearing. The air-conduction hearing aid is preferred to the bone-conduction aid except in cases of active discharging ears. A dry perforation and even postoperated endaural radical cavities are no contraindication to wearing an ear mold. If the two ears are equally impaired, two ear molds can be made and the aid worn in either ear, changing off from time to time. When one ear is more impaired than the other, if the less-involved gives serviceable hearing and the more greatly impaired ear can be brought to a serviceable level, then the aid should be worn in the worse ear. The patient must be told to use the aid constantly and should not give up until he has used it for at least six months. In patients with only small amounts of residual hearing, the hearing aid may be a very valuable adjunct to lipreading. The purpose of this office procedure is to determine whether the patient will benefit by the use of a hearing aid and how much. The patient also feels that his problem is being handled with impartiality and understanding by his otologist rather than by a businessman exclusively.

Berger and Sachs[52] report a case of severe bilateral perception deafness associated with the prophylactic use of tetanus antitoxin: This condition occurred one week after the injection and was associated with generalized giant urticaria. The vestibular system was uninvolved. Two other cases have been previously reported by other writers, both occurring some days after the injection, associated with or following a severe serum reaction. These other two cases also revealed definite labyrinthine involvement, with either a loss or a marked diminution of response to vestibular stimulation. Two other cases of perceptive deafness have been reported following intensive therapy of tetanus with tetanus antitoxin, associated with serum sickness.

The problem presents itself as to what can be done when a patient with serum sickness shows a tendency toward tinnitus, vertigo, and impairment of hearing. The therapeutic armamentarium should include the immediate use of epinephrine, the antihistamines, cortisone, histamine, etc. These measures must be used as the symptoms present themselves.

Macfarlan[53] presents a complete list of the studies and research work done in the United States in 1952. The names and addresses of researchers and organizations carrying out this work are given so that anyone interested may inquire for further information.

An auditory image theory of hearing is advanced by M. Kraus.[54] Various theories of sound perception are discussed. At present the single localization theories (Helmholz, von Békésy) are dominant. Multiple localization theories, e. g., Ewald's auditory image theory, are ignored. Many anatomical and physiological aspects are inadequately explained by the single localization theory. Unsolved problems lie in the recognition of intervals and the fact that few persons have absolute pitch, which should offer no difficulty from the point of view of the single localization theory. The presumption that the basilar membrane is set in motion as a whole is a better explanation. These considerations do not contradict the single localization theory but complete and perfect it.

Stacy R. Guild [55] evaluates the effect on hearing by partial section of the eighth cranial nerve in man, as done by Dr. Dandy in 700 cases of Ménière's disease for the relief of vertigo. His tests were made before and a week to 10 days after the surgical procedure, which consisted of partial resection (one-half to seven-eighths) of the nerve, attempting as far as possible to limit it to the vestibular portion if recognizable. In theory one would expect tone islands or tone gaps. These were not present in a single instance. The changes in hearing were not explainable on any theory of hearing known to the writer. He says further that he himself has no satisfactory concept of how physical sound gives rise to sensations of hearing.

The above observations amount to experiments on man and must be taken into consideration, along with other pertinent facts of record, in any theory of how we hear.

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Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Roland Russell, M.D., *President*

Stanton A. Friedberg, M.D., *Secretary*

Monthly Meeting, Dec. 7, 1953

Abstracts of Papers

Otologist's Role in the Industrial Hearing Conservation Program. Dr. Meyer S. Fox, Milwaukee.

Otologists who are interested in hearing conservation programs in industry may cooperate with and often supervise industrial nurses and physicians and safety personnel and contribute their share toward the accomplishment of a sound hearing-conservation program, the purposes of which are (1) to conserve the hearing of workers; (2) to prevent unnecessary economic loss to employers, and (3) to collect and evaluate research findings and data in order to increase theoretical knowledge and practical effectiveness in this field.

Because of his special training and clinical experience, the otologist can offer realistic advice on (1) the tailoring of a practical program to meet the needs of a particular plant; (2) selection of a suitable room for conducting hearing tests; (3) selection of an audiometer and checks of its performance; (4) instruction and guidance of plant personnel in conducting hearing tests; (5) evaluation of preemployment and periodic hearing tests; (6) placement of workers with impaired hearing; (7) selection, fitting, and use of ear-protective devices; (8) the susceptibility of individual workers to occupational hearing loss, and (9) the detection of willful exaggeration of either hearing ability or hearing loss on the part of workers.

As the hearing conservation program is installed and begins to take effect, the average industrial concern will experience a pronounced decrease in both subjective complaints from workers and the number of hearing-loss claims filed with the Industrial Commission. The otologist can participate in the medicolegal evaluation of such claims and differentiate between noise-induced hearing loss and losses attributable to other causes. He may be called upon to render expert opinions on such thorny medicolegal issues as the following:

At what level of sound intensity and over what period of exposure does hearing damage take place?

What was the status of the employee's hearing when he began work for a particular employer?

What tests should be used for assessing the hearing loss?

What formulae should be used to evaluate hearing-test results in terms of percentage of disability?

Which of several different hearing-test results obtained by several doctors on different days should be accepted as valid?

Are some workers more susceptible to noise-induced hearing loss than others?

How long after his removal from intensely noisy working conditions can a worker's deafness be judged to be permanent?

Often the known facts in a particular case allow only a partial or provisional answer to these questions. The need for research and the paths in which it should be directed are plain. Already, results of hearing studies coupled with medical and industrial histories have been obtained on thousands of workers in the Wisconsin area. Together with similar data collected in other states, these statistical studies are beginning to fill in the gaps in our knowledge and to make speculation about the facts of hearing loss in industry unnecessary. It is hoped that more

otologists will interest themselves in the efforts of industry, labor groups, and others to overcome the hearing-loss hazard and that through the continued cooperation of the various members of the hearing conservation team the problem will be overcome.

DISCUSSION

DR. H. G. KOBRAK: We are indebted to Dr. Fox for this interesting discussion of the problems of industrial noise. We are more indebted to him because of the decision he made a number of years ago to study the problem in detail. Under considerable difficulty and with great personal sacrifice he started an active audiometric study in industry and thus provides the otological profession with much-needed data for further studies. He has made it clear that we are still in the initial phase of the problem. The industrial and military causes of ear damage by excessive stimulation are still growing, and the legal problems are attaining an increased momentum.

The otologist has two problems to solve. The practicing otologist is being asked to examine and to interpret the hearing of the individual patient, and the otological profession must provide answers to the generalized questions which Dr. Fox has named.

So far as examination of the patient is concerned, much discussion has been centered on the question of pure-tone audiometry *vs.* speech audiometry. The scope of usefulness of each method has not always been respected fully. The pure-tone audiogram is an excellent diagnostic method. It permits the diagnosis of the hearing impairment; it indicates the site of the lesion (the conducting mechanism in the end-organ or retrolabyrinthine). But it is a completely different problem to assess the quantity of damage for medicolegal purposes. The patient who demands compensation is not suing because he has a pathologic, absolute pure-tone threshold audiogram; he is concerned because he cannot understand conversation. Would it not be more direct to test the perception of the spoken word? Pure-tone audiometry is not easily translated and evaluated into speech preformance. We have several formulas to assess and to interpret the curves (American Medical Association, 0.8, Fowler, Kinney, Veterans Administration, etc.). But it would seem to be a better investment to direct our efforts to the development of speech audiometry than to the designing of new calculations and arguing about the interpretations of the pure-tone audiogram.

As to the general questions which the otological profession must answer, many otologists have had the experience in court proceedings of having questions such as "What is a safe noise level?" and "How long may an exposure be tolerated before it becomes damaging?" posed to them. One cannot assess the damaging effect of a noise level without considering the time of exposure and the legnth of the recovery period. As biologists, we must insist that legal minds do not succeed in tying a strait jacket around biologic problems.

A word with regard to predetermination of susceptibility of some workers to excessive noise. It was the opinion of otologists at the International Congress in 1949 that fatiguing tests of short duration, as recommended by Peyser, Kriml, Wilson, or Theilgand, do not permit differentiation between resistant and hypersensitive ears. It would seem, therefore, that it is not a wise procedure for the otologist to grade applicants. He should not advise management to abstain from hiring a man on the grounds of his performance in the preemployment fatiguing test. However, prevention of noise damage is possible if the applicant has a preemployment audiogram and repeated audiometric examinations thereafter; the susceptible workman with early damage and poor recovery powers can be weeded out. He can be detected before serious permanent damage occurs in his cochlea. Inasmuch as there is no treatment known for cochlear pathology once it has occurred, the importance of early diagnosis is all important. The value of audiometric examinations as preemployment and as follow-up studies must be stressed. The worker with above-average noise sensitivity can be detected before his training and experience become so advanced that management may be unwilling to transfer him to a different job. This is the positive answer otology can give to industry.

No single man, group, or profession can study fully or solve the problem of acoustic trauma. Teamwork is essential. Two years ago the University of Michigan organized an intensive study course for all those interested in the industrial noise problem. For three days an intensive study and exchange of opinions brought out all important aspects. Many questions remained

unanswered, and many fields for further study were pointed out, among which was the need for facts and for field work. This is the great value of Dr. Fox's work. By giving us the figures and relating his experience in the field, he has provided the basis for evaluation and planning. His work is an important cog in the machinery which has been set up to fight the dangers and the damages of acoustic trauma.

DR. ALFRED LEWY: Dr. Fox has presented a subject that should be of great interest to the otological profession as well as to industry and undoubtedly will be to insurance circles. It has attracted much interest in the Scandinavian literature, and even little Finland has a new law, since 1953, which requires preoccupational examination of the hearing of workers in noisy industries and periodic examinations thereafter and contains clauses concerning the structure and mechanical control of noise in factories, shipyards, etc. It is quite probable that with Workmen's Compensation laws in this country covering damage to the hearing the insurance companies will see to it that Dr. Fox's suggestions are considered.

In the state of Illinois only total deafness is compensable—an unfair situation. Moreover, arbitrators on the Industrial Board are not agreed as to what constitutes "total deafness," i. e., whether it means total for industrial purposes, insofar as it limits a man's chances for a job and his safety on it, or total in the absolute sense. So far as I know, the Supreme Court has not yet ruled on such a case.

Wide-Field Laryngectomy. (Motion picture.) DR. WALTER P. WORK, San Francisco.

This is a motion picture illustrating the detailed techniques in wide-field laryngectomy. The approach to the surgery of this area of the neck is stressed according to facial planes and pertinent anatomic structures. These are worked out on fresh cadaver material, and the motion picture illustrates the technique and procedure of dissection. In addition, a collar type of incision used by the author is shown. Two sections of the picture bring out the operation as performed on a fresh cadaver and, secondly, the actual operating-room procedure on the living. The film is accompanied by an explanatory sound track.

DISCUSSION

DR. BURTON J. SOBOROFF: The movie which we have seen shows a technique of wide-field laryngectomy easily applicable in all those cases in which laryngectomy is indicated. We need not discuss the indications and contraindications for laryngectomy, since these have been so well presented on other occasions.

However, all those who have done this operative procedure have had cases in which there has been local recurrence which might have been prevented by wider resection. These recurrences may develop in the region of the thyrohyoid membrane, above the tracheal stoma, or about the tracheal stoma and in the tracheal mucosa. The procedure of wide-field laryngectomy extends the operation from the tracheal area to the level of the base of the tongue, including in dissection the strap muscles and the hyoid bone. Removal of the hyoid bone provides for adequate removal of the entire preepiglottic space into which tumor can so readily extend anteriorly from the larynx. Removal of all the strap muscles in a single block dissection is done in an atempt to remove any tumor which may have extended anteriorly from the larynx into these tissues or through a prelaryngeal node.

Resection of the entire cricoid, together with one or two tracheal rings, provides a wide field of safety below the lesion in the event of subglottic extension, which, in many instances, cannot be seen accurately before surgery. Dr. Work has not presented any additional wider indications for total laryngectomy but has tried to emphasize that in those cases in which laryngectomy is indicated, a wide-field procedure, such as the movie demonstrates, should be done.

DR. MAURICE F. SNITMAN: I had the opportunity to preview both films, and it was quite obvious that the operative procedure on the cadaver was much more instructive and understandable. Color productions of film of surgical procedures on the neck of the living are, as a rule, unsatisfactory, and the same holds true for still and movie reproductions.

It would appear that the term "wide field" was coined for the purpose of drawing attention to a procedure that is contrary to narrow-field laryngectomy. In this presentation the author

does meet an important requirement of neck surgery, that is, adequate exposure and dissection. This basic feature I heartily commend, although I do not favor a few of the technical elements. I am especially critical of the term "fascia," which is so frequently repeated in the description of the procedure. I must emphatically state that true fascia of the neck does not exist. What is alluded to is a false impression of a fascial layer, produced by the condensation (as a result of the surgical dissection) of the loose connective tissue of the neck.

Dr. L. Benno Bernheimer: It was a privilege to see Dr. Work's movie. I am sure that his indication for the procedure is the presence of extrinsic disease. I am still of the opinion that intrinsic noninfiltrating carcinoma responds as well to radiation as to surgery, but, on the other hand, for the type of disease that I assume the author is treating, radiation would be merely palliative. Radical neck surgery is, of course, not new. Radical neck dissection and laryngectomy with wide exposures were performed many years ago, but the mortality was so high that such procedures were abandoned. The advent of chemotherapy and the antibiotics has altered this picture, so that today almost nothing is considered anatomically inoperable.

Dr. Irwin D. Horwitz: The procedure just demonstrated was for carcinoma of the extrinsic larynx or hypopharynx. Any criticism of this should be based on the fact that the operation was not extensive enough not on any esthetic viewpoint that it is too mutilating Tumors of this area metastasize early, and most radiologists will concede that they cannot successfully treat metastatic tumors. The best results claimed are between 10 and 12% for five-year cure rates. The suggestion of Dr. Bernheimer that x-ray and irradiation be used does not follow the dictates of practical therapy. A treatment consisting of one-half surgery and one-half radiation adds up to a therapeutic zero. In past years wide extensive surgery has offered the promise of a better salvage ratio and, equally important, an earlier rehabilitation.

Inner-Ear Deafness Due to Measles. Dr. John R. Lindsay.

Loss of hearing due to measles has commonly occurred in two ways: the result of bacterial middle-ear infection and an acoustic neuritis due to the measles virus. Antibiotics have been effective in controlling the middle-ear infections but not the acoustic neuritis or inner-ear deafness due to the measles virus.

Several surveys have indicated that inner-ear deafness due to measles accounts for 3 to 5% of acquired deaf-mutism. Unilateral deafness and bilateral hearing impairment are also reported to be not infrequent.

Reports on the pathology of uncomplicated acoustic neuritis due to the measles virus have been extremely rare.

The inner-ear changes in a child who died of complicating myocarditis several weeks following measles are presented. Both ears showed changes in the organ of Corti, tectorial membrane, and stria vascularis, more marked in the basal turn. There was nerve and ganglion degeneration in the basal turn. The utricles, saccules, and cristae of the semicircular canals were also involved. Lesions were evident in the stria vascularis and utricle, which may represent the basic pathologic lesions due to the measles virus.

DISCUSSION

Dr. Elmer W. Hagens: In 1930 several of us reported on the examination of more than 5,000 children in schools for the deaf. Of this group, about 2,000 had acquired deafness; 172 children, or 8.5%, had become deaf after measles. Considering the measles cases alone, we found that 51 had an associated otitis media, while 121 had become deaf without otitis media. Both groups were divided into total and partial deafness, and the vestibular reactions were recorded on all. As would be expected, the cases of total deafness without a history of otitis media showed complete loss of vesibular reactions in 50%. On the other hand, in total deafness associated with otitis media, 30% had no vestibular reactions. The cases of partial deafness had a much lower percentage of cases without vesibular reaction. In the report we stated that it could be assumed that a toxic neuritis was no doubt responsible for the severe deafness in the two groups. The lack of vestibular response in many of these children also supported this view.

The child whom Dr. Lindsay referred to from a previous article of mine was 14 years of age and had developed measles one year before her death. Knowing her family doctor, I was

able to determine that she had been practically totally deaf for that year. Death occurred following a new respiratory infection, with intracranial complications. I was able to obtain only the right temporal bone, as the pathologist retained the other side.

Study of the microscopic sections showed no evidence of labyrinthitis, but there was complete absence of the organ of Corti and a considerable destruction of the vestibular end-organs. The spiral ganglion cells were destroyed in great part, while Scarpa's ganglion cells were fairly well preserved. I believe that this case falls into the category discussed by Dr. Lindsay, as there was no labyrinthitis to obscure the picture. Such reports as Dr. Lindsay's will help to clarify what happens to the ear in cases of toxic neuritis.

Monthly Meeting, Jan. 4, 1954

Abstracts of Papers

Management of Ménière's Syndrome. Dr. Frank D. Lathrop, Boston.

A universally satisfactory method of managing patients afflicted with Ménière's syndrome apparently has failed to be realized even though the symptomatology and pathology of this syndrome are well established. This is attested to by the numerous and variable forms of therapy for this malady that have appeared in the literature during the past decade. It is not my purpose to discuss the pros and cons of these various forms of treatment, since I intend to offer for consideration a program of management for Ménière's syndrome that has proved satisfactory in my hands.

In order for any method of treatment to be effective it is necessary that the correct diagnosis be established. It should be recognized that the triad of symptoms characterizing Ménière's syndrome (deafness, tinnitus, and vertigo) may be the manifestation of many diseases. The diagnosis of Ménière's disease should be made only when no other explanation can be found for these symptoms. Patients with typical Ménière's syndrome complain of tinnitus and severe attacks of labyrinthine vertigo associated with bilateral nerve deafness, which is usually more profound in one ear. The diagnosis cannot be substantiated on the basis of a single, isolated attack of labyrinthine vertigo even though it may be accompanied by deafness and tinnitus, since, by definition, the vertiginous attacks must be recurrent. The hearing early in the course of the disease may fluctuate to a considerable degree, with the lower frequencies most affected, but as the disease progresses, the hearing loss becomes more profound, fluctuates little, and higher frequencies are most affected.

Approximately 85% of the patients whom I diagnose as having Ménière's syndrome respond satisfactorily to the following medical regimen. This consists of a diet low in sodium content and the administration of ammonium chloride to facilitate the elimination of unnecessary sodium from the body. The patient is instructed concerning a diet which permits him to eat most of the food he desires as long as it is not salted at the table. Articles of food that are cured with salt, such as ham and bacon, are prohibited, as are flavoring agents, such as catsup, mustard, Worcestershire sauce, and so forth, since they contain excessive quantities of salt for the dietary purpose. Pepper, olive oil, and vinegar may be utilized as desired to flavor the food. Ordinary bread and butter in normal quantities may be used. This diet can be followed equally well in the home or the restaurant and thus eliminates to a large degree the impracticability of following a strict dietary regimen when the patient is away from home.

Ammonium chloride is utilized as an enteric-coated tablet. The initial dosage is 2 gm. three times a day with meals for three days, omitted for two days and repeated at these intervals for an indefinite period. The patient is instructed to take the tablets spaced throughout his meal. If he is in the habit of eating a small breakfast, two tablets may be taken then and five tablets with dinner and supper, or if both breakfast and lunch are small meals, two tablets are taken with each of these meals, four with supper and two tablets between breakfast and lunch and lunch and supper. If this manner of administration is followed, the incidence of gastric irritation is reduced to a minimum.

The success of this form of treatment is augmented if the patient is made aware of the nature of his illness and thoroughly understands the rationale of the medical regimen, as only

through this knowledge can he actively participate in the mangement of the therapy, just as does the diabetic. He must be seen at periodic intervals in order to observe his progress, make necessary alterations and additions to the medical regimen, and strengthen the relationship between patient and physician, so that questions may be answered reasonably and assurance given that something else can be done to relieve the dizziness if this treatment fails. Since many patients with Ménière's syndrome are either in the older age group with additional symptoms secondary to arteriosclerosis or are nervous, nicotinic acid, mild sedation, or both are often helpful in further effecting their rehabilitation.

In the event that the patient fails to obtain relief from the paroxysmal attacks of vertigo from any form of medical therapy that may be employed, recourse must be made to some form of surgical procedure. For this purpose I have found the Cawthorne operation most satisfactory. This consists of sufficient removal of the cellular structure of the mastoid process to permit avulsion of the membranous semicircular canal through a small fistula created in the bony horizontal semicircular canal. This is a simple procedure as compared with intracranial section of the eighth cranial nerve and, in my opinion, is more satisfactory in effecting rehabilitation of the patient. It should be emphasized, however, that if the medical management of Ménière's syndrome is assiduously carried out, the necessity for surgical intervention will be infrequent.

DISCUSSION

Dr. John Ballenger: I was surprised that Dr. Lathrop did not make more mention of the use of autonomic blocking agents, particularly of the cholinergic nerves. I assume that he employed dimenhydrinate (Dramamine) for that purpose, but there are others, such as methantheline (Banthine) bromide or tetraethyl ammonium, which would seem to be more efficient over a longer period; 50 mg. of methantheline three times a day, in my more limited experience, has been quite helpful.

I should also like to inquire about the fluid intake. At one point he suggested a high fluid intake if there was a reaction to the use of ammonium chloride. Except for this, it would seem to me that a low fluid intake would be better advice to the patient.

Dr. Alfred Lewy: We are indebted to Dr. Lathrop for a most detailed description of Ménière's disease, to which I cannot add much. I should like to suggest that the audiogram would be effective here in helping to indicate in which ear surgery should be done, in case surgery is contemplated. An erratic audiogram or a changeable one would lead one to suspect Ménière's disease. Also, I would be suspicious, as he is, of a patient who reacts more violently to a caloric test than ordinarily; in that case I would suspect psychoneurosis. Of course the patient with Ménière's disease might also develop psychoneurosis. The old treatment with pilocarpine is probably effective by a dehydration effect. I have used pilocarpine before any other definite treatment was known, but the success of that requires hospitalization.

I should like to call attention to a drug which was not mentioned by the essayist, that is, pyridoxine (vitamin B_6), and to ask what his experience has been with this. In 1947 Dr. Fox and I published a report on some 87 cases of vertigo, of which 17 disappeared from our care and were considered failures. Of the 87 cases, 47 were diagnosed by us as Ménière's disease, the others were all vertigo of unknown etiology. Of the latter group many were helped by the use of pyridoxine, and 75% of those diagnosed as Ménière's disease were benefited. Since then I have definitely increased the dosage; we formerly gave 50 mg. t.i.d. We now give 50 mg. ordinarily and in more severe cases 100 mg. In the very severe cases intramuscular or intravenous injections are given.

I do not know the pharmacology of pyridoxine. It has been used for the nausea of pregnancy and does not always work there. It has also been used successfully for treatment of agranulocytosis, and some neurologists have reported it effective in relieving the spastic symptoms of multiple sclerosis. I do not know how it works and wish that someone would tell me. It is not very successful in motion sickness; dimenhydrinate is probably better.

Dr. John R. Lindsay: I should like to congratulate Dr. Lathrop on his very careful and clear description of the symptoms of Ménière's disease and the attention he pays to making an accurate diagnosis. It is very important that this be done, since even after many years in which Ménière's disease has been discussed a great deal, there is still some confusion about what constitutes true Ménière's disease.

One point that is important in the general management of the disease, and which is exemplified by the detail with which Dr. Lathrop has described his medical management, is the care taken to regulate the patient's daily life in all its aspects. Whether it is mainly the psychic effect of his knowledge that you are taking a great deal of interest in his case, I am not sure, but I suspect that the psychic aspect has much to do with it.

I agree with Dr. Lathrop that there is no point in using coagulation in the labyrinth. I have had no accidents with it, but I have seen one case of total facial paralysis following coagulation. I have also seen one case recently which, followed the Cawthrone operation, had a recurrence; repeated attacks came back, and in that case I opened both the horizontal and the superior semicircular canals, opened the vestibule widely, and destroyed its contents. I see no reason why that should not be done in the first place; rather than just the horizontal canal, why not do a more thorough destructive job on the contents of the vestibule.

Dr. Sherman Shapiro: Our distinguished speaker has given an excellent summary of the subject, and I might say that the drugs he mentioned, ammonium chloride and nicotinic acid, are precisely those upon which I have relied for a good many years. There is one point I should like to add from the standpoint of diagnosis and that involves the use of something of which I believe Dr. Lindsay and I are perhaps the chief protagonists in this vicinity, namely, the Frenzel glasses. In most cases of frank Ménière's disease, nystagmus can be seen quite readily, but after the acute attack is over many patients may feel rather well; however, while nothing is seen by the unaided eye, when the glasses are put on they exhibit a horizontal nystagmus, which, of course, enables the physician to say at once that the condition is not psychic. This, also by laws of labyrinthine irritation and destruction, sometimes helps to differentiate between the two sides in cases in which this is in doubt. Many of these patients have just a slight dizziness, as Dr. Lathrop mentioned, and it is a satisfaction to know that they are not just imagining it.

I should like to ask Dr. Lathrop what his experience has been with potassium instead of ammonium chloride.

Dr. Robert Lewy: I should like to ask Dr. Lathrop how he differentiates with relative certainty the fact that he is not dealing with a supravestibular type of vertigo. Certainly we have all had to deal with this from time to time, and any destructive operation on the vestibule under those circumstances would have a greater tendency in the direction of failure, I am sure. Does he use the recruitment test, among other things, in helping to establish the diagnosis? Does he make an allergic evaluation of the patient? I have had some good results in finding some specific allergens in the form of food which would precipitate a Ménière's attack, and removal of the offending allergen, of course, solves the problem.

In terms of surgical intervention, my colleagues and I have been working for seven years in a large public institution which receives difficult cases from all over the United States. We have not had one case which, by our standards and in our opinion, has required a destructive operation of the labyrinth. It has always puzzled me as to whether our standards are different or whether, by virtue of reputation or for some other reason, those cases in which surgery is necessary get into other hands. Prior to the work of Passe and Seymour, in which they cut the thoracic anterior gray rami (first they took out the stellate ganglion, then later the anterior gray rami in the upper thoracic region), the only surgery done was one or another type of destructive surgery. Using their work as a basis, Hoagland proceeded to do repeated stellate ganglion blocks and claims to have had some very satisfactory results and to have avoided surgery. I have not had much experience with it, but it would seem that if he is right, a patient should be subjected to a trial repeated stellate ganglion block and have a destructive operation on the cochlea only if this fails.

Dr. Elio Forjatto: I should like to mention some work that has been done in Italy on surgical procedures for the partial destruction of the labyrinth, with suppression of the vestibular function without deterioration of the cochlear function. As we know, the Cawthorne operation involves complete suppression of the hearing function in the ear operated upon. Two surgical procedures have been studied in Italy. At the University of Turin a new method has been carried out in animals and in man. This consists of a fenestration operation with introduction of a fibrin sponge in the semicircular canal in order to determine the formation of

fibrotic tissue. The histology of successive changes has been studied. Animals show a normal cochlear function after destruction of the vestibular labyrinth. In man, however, the hearing function deteriorates until there is complete loss. Therefore the value of this procedure will be seen only in the future. At the University of Padua, Professor Arslan destroys the function of the vestibular apparatus by direct application of a bundle high-frequency sound waves to a semicircular canal after an extensive mastoidectomy. A special apparatus is employed, and particular care is needed to avoid facial palsy. In all cases the hearing remained the same as before the operation. The results have been controlled several months after operation.

DR. HENRY B. PERLMAN: In the hope of obtaining some information about the pathogenesis of this disease and to evaluate the commonly employed low salt diet regimen, we recently studied a group of patients with active Ménière's disease in the hospital with the cooperation of Dr. Goldinger, of the Medical Department.

Ordinarily it is very difficult to change the body electrolyte balance by dietary means. In these hospitalized patients we were able to produce a small but measurable reduction in the serum sodium. This was accomplished after a week or two on a 300-mg. sodium diet, with the administration of mercaptomerin (Thiomerin) sodium and of ammonium chloride and, sometimes, intravenous aminophylline. These patients had active Ménière's disease, as manifest by measurable fluctuating hearing loss and recurrent attacks of vertigo. Their clinical state was not changed during the period when there was a measurable reduction in this serum sodium. Auditory threshold curves continued to fluctuate as before, and two patients had a typical acute violent attack of vestibular vertigo and nystagmus.

We then did a converse experiment with these patients—that of deliberately raising their serum sodium. This was accomplished by giving them desoxycorticosterone, additional sodium chloride in the diet, and restricting their fluid intake. A measurable increase in the serum sodium was obtained. Strangely enough, the patients did not get worse under the regimen. Their hearing did not deteriorate, nor did they experience attacks of vertigo. Instead, in two patients there was a return of the previously reduced auditory function to a practically normal level. It is of interest that despite all our investigations and astute clinical observations, the pathogenesis of this disease is obscure.

DR. FRANK D. LATHROP: I do not know the answer to this particular problem. Certainly it is very difficult to demonstrate any particular changes so far as electrolyte balance is concerned, but the fact remains that the program I have offered to you will give good results if it is followed as assiduously as I suggest.

This brings up a very delicate question. Are these patients benefited because of the medical regimen or because they have confidence in the doctor? I do not know, but it makes no difference to me, since the patient is happy and is able to work, which he could not do before. You cannot just give these patients something to do, you must follow them along. Sometimes I find that after I have seen them two or three times that the diagnosis of Ménière's syndrome is erroneous, and I change the program of treatment. But the point I wish to emphasize is that the patient cannot be treated offhandedly. He must be made to realize that he has someone to whom to turn in case he has trouble and that his physician is willing to help when he is in trouble.

I learned long ago that if I desired to become proficient in the management of a disease it was necessary for me to learn how to handle all the details of one or two forms of therapy. I do not believe that there is a man alive who can do neurosurgery, otolaryngology, ophthalmology, and general surgery as well as he can an individual specialty.

I believe that the use of methantheline, which Dr. Ballenger mentioned, might be effective. I have never used it because I have found effective measures of relief for my patients without the use of methantheline. It may be a good drug, but I have learned how to use others, and I stick to them.

With respect to the management of an acute attack, and this will also answer Dr. Lewy's question about stellate blocks, I have found that in an acute attack a stellate block with 1% procaine apparently does relieve the dizziness. I do not believe, however, that repeated blocks of the stellate ganglion constitute a rational form of management for Ménière's disease. I have yet to see a case of a cerebellar lesion or supratentorial lesion which has produced dizziness

that resembles that of Ménière's syndrome. Again I wish to emphasize that between the attacks of vertigo the patient may be entirely asymptomatic except for tinnius and deafness, but the patient who has a brain tumor and vertigo is never free from a sensation of unsteadiness.

So far as the fenestration operation is concerned or the plugging of the fenestrated horizontal semicircular canal with an absolute gelatin sponge (Gelfoam), essentially the same thing was employed some years ago by Dr. Lindsay, without permanent beneficial results being obtained.

The point Dr. Shapiro brought out with respect to Frenzel glasses is very good. Strangely enough, an occasional patient will not respond to ammonium chloride but will respond to potassium chloride and a low salt diet.

With respect to fluid intake, at no time have I said that the patient should have a high fluid intake. I ask him to continue his normal fluid intake. Many patients automatically cut down on fluid intake when they start a salt free diet. Such patients frequently become constipated, and I ask them to increase the fluid intake but not to make it a high fluid intake.

Dr. Lindsay asked whether I used streptomycin. I have not for the reasons, again, that I have been able to rehabilitate the majority of my patients with the therapy I have described. Thus far I have had no failures from surgical intervention.

Some Problems in Antibiotic Therapy. DR. HARRY F. DOWLING.

The antibiotic-resistant strains of bacteria which are a problem in otolaryngology are almost entirely staphylococci. These, however, constitute a problem of ever-increasing importance. We have found that resistant strains may appear as a result of (1) treatment of a patient with a staphylococcic infection; (2) spread from a patient who has been so treated, or (3) spread from contacts of patients who have been treated with the antibiotic, specifically hospital personnel. The emergence of resistant strains may be delayed by the use of combinations of antibiotics in some instances but can be prevented only by rapid termination of the infection and discontinuance of the administration of the antibiotic. The spread of resistant strains can be prevented by proper isolation technique and by the development, in new hosts, of resistance against infection.

The wholesale use of antibiotics for upper respiratory infections is to be condemned because it increases the frequency of hypersensitivity reactions and accelerates the emergence of resistant strains.

Among the new drugs, erythromycin and carbomycin are of some value for infections caused by Gram-positive cocci, while tetracycline (Achromycin) is an analogue of chlortetracycline (Aureomycin) and oxytetracycline (Terramycin) which gives fewer gastrointestinal reactions, is more stable, and penetrates into the spinal fluid more readily than chlortetracycline or oxytetracycline.

DISCUSSION

DR. ROBERT HENNER: What is your feeling about the routine use of pencillin in preoperative cases in which there is no infection?

DR. SHERMAN SHAFIRO: Do you know how the development of resistant strains is affected by the combined use of such antibiotics as pencillin with the sulfonamides?

DR. ROLAND RUSSELL: What about the local use of antibiotics?

DR. STANTON A. FRIEDBERG: I wonder if you would tell us of any experiences you have had in which antibiotics, through a toxic effect, were able to mimic a severe infection and thus be misleading.

DR. SAMUEL J. PEARLMAN: What do you think of combinations of antibiotics and antihistaminics?

DR. HARRY F. DOWLING: With regard to the prophylactic use of penicillin preoperatively, I feel that there should be some good controlled series before such measures are carried out. It is strange that there is little in the literature concerning such studies on the prophylactic use of penicillin. One report made by a thoracic surgeon showed that the incidence of empyema was lower in patients who received penicillin routinely before operations on the lung. However, until we know more about it, I feel that antibiotics should be used prophylactically only when operation is performed in an infected area or when there is a threat of infection. If that rule were followed, we would not have so many patients becoming sensitive to the antibiotics.

There is no evidence that a combination of penicillin and one of the sulfonamides will delay development of resistant strains. The sulfonamides are so much less active than the antibiotics that they do not add much. The percentage of resistant strains would be about the same. That is particularly true of the Staphylococcus.

There is little reason for the local use of pencillin or any other antibiotics. Certainly, in my opinion, troches or lozenges should never be used. They give a concentration on the surface of the mucous membrane of the mouth, but that is not where the bacteria are. The use of the antibiotics in the sinuses is another matter, because the material remains there for a certain lengh of time and can penetrate the membranes. I believe that penicillin should be employed only occasionally on the skin (although it is being used frequently), and again we may get sensitivity reactions from such applications.

Sensitivity reactions can mimic infection, particularly in the acute attacks with fever, skin reactions, joint pains, and swelling. A careful history can usually elicit the cause, and the antibiotics can be stopped and the reaction controlled.

I prefer to reserve the use of antihistaminics until the reaction is noted, because of their masking qualities. In a severe reaction they have little effect and will not prevent an acute anaphylactic attack.

Abstracts from Current Literature

Ear

THE OTOLOGIST LOOKS AT THE INDUSTRIAL NOISE PROBLEM. M. S. FOX, Eye, Ear, Nose & Throat Month. **32**:433 (Aug.) 1953.

Otologists are being consulted with increasing frequency by those vitally affected by the industrial noise problem. This interest has risen sharply in the past few years partly as a result of damage suits brought before industrial commissions and courts and partly from a humane desire to conserve the hearing of workers exposed to hazardously high noise levels. The problems of noise in industry are extensive and complicated due to the fact that they involve many complex acoustic, medical, and legal questions. Therefore the otologist who is to give information and advice must be properly informed not only concerning the otological but also the physical and legal aspects of the problem. There are many factors which affect the degree and extent of hearing inpairment found in each case of noise deafness. There is general agreement that susceptible persons exposed to intense noise levels for a sufficiently long period of time will sustain a loss of hearing. There are four important factors involved: intensity of the noise, duration of exposure, frequency spectrum of the noise, and individual susceptibility.

It is necessary to have an objective measure of sound, either by the sound level meter or the wave analyzer. The latter provides a more accurate and informative analysis of sound. For sudden impact sounds which generate swift powerful pulses a new method which uses the cathode ray oscilloscope and high-speed photography has been devised. In the same person each ear may vary in ability to withstand noise if one of the ears had a preexisting conductive lesion. The person with a preexisting conductive lesion is especially adapted to work in noisy areas, but the reverse is true of many persons with preexisting perceptive losses. In several industrial programs in which audiometric tests are routinely performed on all applicants, all workers showing any appreciable hearing loss are rejected, probably due to the absence of complete otological examinations.

Before making a diagnosis of an occupational hearing loss, the otologist should satisfy himself that the worker has the nerve-type hearing impairment characteristic of acoustic trauma, that sound-level measurements of the working area reveal intensities sufficiently great to have caused hearing loss, and that there has been sufficient exposure time. The hearing loss produced by prolonged exposure to loud noise may be considered as permanent six months following complete removal of the worker from the area of the loud noise.

From a medicolegal standpoint, it is necessary for the otologist to be objective, scientific, and impartial. There is no place for partisan attitudes in the final determination of awards for occupational hearing losses. This lies within the jurisdiction of the authorized courts. It should be founded on the knowledge of all the factors involved in industrial deafness, and this is possible only through cooperation of labor, management, and the medical profession.

JENNES, Waterbury, Conn.

AUDIOMETRIC TESTING OF HEARING OF SCHOOL CHILDREN. SAMUEL M. WISHIK and ELIZABETH R. KRAMM, J. Speech & Hearing Disorders **18**:360 (Dec.) 1953.

Hearing tests were made on 1,726 children in the fifth and sixth grades in the Reading School District, Reading, Pa. All had been routinely tested in the first, third, and fifth grades. In the first six grades 6.5% of the children given an audiometric test failed one or more times. The finding of new cases amply justifies a program of biennial testing through the first six grades. A significant number of children have impaired hearing for periods of less than two years' but of more than several weeks' duration.

PALMER, Wichita, Kan.

MASKING AND FATIGUE EFFECTS OF WHITE NOISE IN CONNECTION WITH SPEECH TESTS IN OTOSCLEROTIC SUBJECTS. CARLO SAMBATARO and GIULIO PESTALOZZA, Laryngoscope **63**:732 (Aug.) 1953.

Otosclerotic patients were examined in a quiet room with the use of two loud speakers—one emitting white noise and one logotomes. Articulation tests were made with and without the masking effect and also after fatigue (60 db. for 15 minutes). For low intensities of white noise, the threshold shift in otosclerotics was less than normal; for higher intensities it was practically normal. Also, after an otosclerotic is subjected to fatigue, the articulation curve does not improve as with the normal subject.

HITSCHLER, Philadelphia.

MANAGEMENT OF CONGENITAL EAR-CANAL ATRESIA. HOWARD P. HOUSE, Laryngoscope **63**:916 (Dec.) 1953.

The difficult problem of congenital ear-canal atresia is clearly discussed. The operation to form a new canal and restore hearing is one of the most difficult in otology, particularly if sclerosis is present, and should be undertaken only after careful study.

If the condition is unilateral and there is hearing in the other ear, it is doubtful that the operation should be done. Also the presence of good cochlear function must be determined before operative procedures are undertaken.

HITSCHLER, Philadelphia.

A HELMET-HELD BONE-CONDUCTION VIBRATOR. J. D. HARRIS, H. L. HAINES, and G. K. MEYERS, Laryngoscope **63**:998 (Oct.) 1953.

It is observed that bone-conduction audiometry can be as accurate or more accurate than air-conduction audiometry. Hand-held bone-conduction vibrators have certain disadvantages, mainly the variation that may occur in their position on the skull and the force with which they are held against the skull. A helmet-like apparatus which eliminates these faults is described. Measurements were made with or without the apparatus. Less deviation in readings is obtained with the use of the apparatus, but the difference is a few decibels. Differences in pressure against the skull produce variations in the readings only in the low tones. A force between 200 and 400 gm. should be used to avoid this. It is concluded that only relatively small deviations in bone-conduction readings are found, even with rather wide variations in the mode of applying the sound.

HITSCHLER, Philadelphia.

PRELIMINARY REPORT OF A NEW METHOD FOR THE EXAMINATION OF NYSTAGMUS THROUGH TELEVISION. JOSEPH A. SULLIVAN, WALTER H. JOHNSON, and W. E. HODGES, Laryngoscope **63**:1193 (Dec.) 1953.

Nystagmus was photographed by a television camera. The image was enlarged and transmitted to a television screen. A large image such as this can easily be observed, measured, and, if so desired, photographed.

HITSCHLER, Philadelphia.

PNEUMATIZATION OF THE MASTOID PROCESS IN RELATION TO OCCUPATIONAL DEAFNESS. K. DOHI, J. Oto-Rhino-Laryng. Soc. Japan **56**:537, 1953.

An observation on the relation between the pneumatization of the mastoid process and the loss of hearing was made on 100 laborers working in a noisy environment (exceeding that of 90 phones) over a period of years in a shipyard.

In a group of 68 laborers who had been employed from 10 to 15 years, it was observed that those with well-developed pneumatization had a slighter loss of hearing than those with undeveloped pneumatization. In a group of 32 laborers who had been employed over 30 years, this relation was not recognized. The same relationship between the pneumatization and deafness was observed between the two ears. Therefore air cells of the mastoid process obviously have a preventive influence against the loss of hearing due to strong, persistent noises.

HARA, Los Angeles.

EXPERIMENTAL STUDY OF PATHOLOGIC CHANGES IN THE EAR CAUSED BY GALVANIZATION. T. OBATA, J. Oto-Rhino-Laryng. Soc. Japan **56**:619, 1953.

Direct current of various strengths (7 to 30 ma.) and various durations (10 to 60 seconds) was sent into a guinea pig's ear via the electrodes inserted into each external ear canal.

Decapitation or intra-vitam fixation was performed while the current was still on, and the auditory organ was histologically examined to establish the effect of the more or less prolonged galvanization.

During the galvanization, nystagmus, trembling movement, or sustained deviation of the eyeball was observed. On histological investigation traumatic changes prevailed over the whole range of the auditory organ, especially on the cathodic side rather than on the anodic. Changes both in sensory and in supporting cells of the cristal and, especially, the maculae and in the ganglion cells were on each side quite characteristic.

The galvanization effect on the anodic side was first manifest as nuclear pyknosis in the sensory cells. Stronger current first produced edematous swelling of the cytoplasm, then swelling of the nuclei toward the surface of the neuroepithelium, along with the swelling of the nuclei, and further pyknosis and displacement of the nuclei toward the surface of the neuroepithelium, along with the swelling of the cytoplasm.

Disturbed arrangement and constriction were the first signs detected in the supporting cells as the result of the galvanization, occurring with the swelling of the sensory cells. On the cathodic side a similar but stronger effect was observed, i. e., disorder in the sensory cells, pyknosis of their nuclei, and disturbance in the supporting cells. These effects appeared in an intensified degree as the strength or the duration of the constant current used in the experiment increased.

HARA, Los Angeles.

Larynx

EVALUATION OF INDIRECT LARYNGOSCOPY WITH SPECIAL REFERENCE TO INDICATIONS. J. I. KEMLER, Eye, Ear, Nose & Throat Month. **32**:87 (Feb.) 1953.

Mirror examination of the larynx at times entails a great deal of difficulty and hardship to both the patient and the doctor. Although some patients may be examined easily, most patients have a gag reflex which must be abolished. This can be accomplished by spraying the pharynx and the soft palate with a local anesthetic agent. Kemler feels that it is very important to anesthetize the frenum and the tip of the tongue as well. This eliminates the tendency of the patient to pull the head backward, making the examination difficult. If the patient keeps his eyelids wide open routinely, it greatly facilitates the examination of the larynx.

Kemler lists the advantages of indirect laryngoscopy. It can be done in the office as a routine practice; every part of the larynx can be visualized, usually without any extra instrumentation. The larynx appears in the natural state without congestion or distortion, and the mobility is unhampered. Biopsy can be performed without delay at the first examination, hospitalization is not required, and there is therefore no loss of time from work.

JENNES, Waterbury, Conn.

TUBERCULOSIS OF THE LARYNX AND ITS TREATMENT WITH STREPTOMYCIN AND HYDRAZINE DERIVATIVES OF ISONICOTINIC ACID. RICHARD R. LEHMANN, Laryngoscope **63**:977 (Oct.) 1953.

Tuberculosis of the larynx is always a secondary disease, usually a complication of far-advanced pulmonary tuberculosis. The gross appearance of the larynx may vary from slight inflammation of one cord to swelling, granulation, ulceration, and notching of the cords. The surrounding areas may be involved.

The incidence of tuberculosis of the larynx has diminished greatly in the last 40 years owing to increased knowledge of the disease and improvement in treatment. Streptomycin therapy has been astonishingly successful. Its effectiveness is enhanced and the side-effects reduced by the use of smaller doses (1 gm. every third day). Aerosol streptomycin is ineffective. Isoniazid (Rimifon) is also an effective drug, particularly in cases in which streptomycin has failed. Case reports are included.

HITSCHLER, Philadelphia.

FUNCTIONAL PROBLEMS CONNECTED WITH BILATERAL RADICAL NECK DISSECTION. ETTORE BOCCA, J. Laryng. & Otol. **67**:567 (Sept.) 1953.

Thirty per cent of laryngectomized patients die of metastatic lymph node invasion. Accordingly, prophylactic bilateral radical neck dissection is indicated whenever an extraglottic cancer invades the larynx, even in the total absence of clinical metastases. In Bocca's series of 500 prophylactic or therapeutic neck dissections, it was shown that in over half the cases metastases were clinically mute but were demonstrated histologically. It is difficult to understand why laryngologists have failed to appreciate the principles of regional surgery and are still arguing over a problem which has long been unanimously settled in other fields of cancer surgery.

The usual objections to bilateral neck dissection are that (1) it may often prove an unnecessary mutilation and (2) severe motor and circulatory disturbances may result. In regard to the former, it is better to perform 50% "white" dissections than to allow the other 50% to develop metastases, the outcome of which is most often fatal. Also, bilateral neck dissection adds nothing to the mutilation unfortunately connected with removal of the larynx. The most serious motor effects from radical neck dissection depend upon the interruption of the spinal nerve, resulting in the inability of the patient to elevate the arm above the level of the shoulder and the development of a lower facial palsy should the dissection extend to the submaxillary space. Both can be avoided by careful dissection. The facial palsy is always partial and tends to recover spontaneously in the course of one year. The circulatory effects arising from the interruption of the greater part of the venous channel to the neck cannot be avoided. However, in Bocca's series of 112 bilateral two-stage neck dissections, with an interval of three to four weeks, no fatalities resulted; there were no permanent ill effects, and in only two cases were immediate circulatory effects of a major gravity observed, and these subsided after a period of only 12 hours. Complete compensation in brain circulation possibly never takes place; nevertheless, cerebral and psychic symptoms are totally absent. Facial edema completely disappears in four to six months, but cyanosis of the skin and mucous membrane may persist. Mobility of the head is not appreciably limited by the absence of both sternomastoid muscles. Pains in the shoulder and upper arm, due to irritation or trauma of branches of the cervical plexus, generally subside in the course of the first year. The formation of an esophageal voice is by no means hindered or delayed.

In conclusion, prophylactic bilateral radical neck dissection in two stages is a safe procedure which allows a systematic preservation of important nerves not possible in therapeutic dissection when the lymph nodes are adherent to the surrounding tissues.

LEJEUNE and NORMANN, New Orleans.

Nose

NASAL SURGERY ON THE ALLERGIC PATIENT. K. L. KRAFT, Eye, Ear, Nose & Throat Month. **32**:582 (Oct.) 1953.

Surgery is indicated for the allergic patient when the allergy is complicated by infection, mechanical obstruction, and other nonallergic problems necessitating operation. In such cases it is as wrong to depend on allergic treatment alone as it would be to treat a purely allergic condition by surgery. A rational evaluation is required. If cases of allergic edema of the nasal mucosa complicated by further obstruction from a badly deformed septum or from a hypertrophied fibrotic turbinate undergo the indicated surgical correction, the improved breathing space thus provided may then be satisfactorily maintained by adequate allergic management, which alone, without surgery, would not have been effective.

The repeated removal of recurrent nasal polyps without allergic management is not rational. Similarly, tonsillectomy and adenoidectomy should not be performed in the allergic child without adequate allergic therapy. Kraft cites a survey by Clein which showed that 27% of the untreated allergic children undergoing tonsillectomy and adenoidectomy had regrowth of lymphoid tissue as compared with an incidence of regrowth of only 3% in those patients having adequate allergic management.

JENNES, Waterbury, Conn.

EVALUATION OF A NEW SPRAY DEVICE FOR INTRANASAL MEDICATION. D. G. ORNSTON, Laryngoscope **63**:739 (Aug.) 1953.

The use of the spray bottle for intranasal medication was tested in various ways to determine its efficiency. Observing the mucosal coverage of sulfathiazole crystals and x-ray studies of a radiopaque medium aided in the evaluation. It was concluded that the spray bottle, although not quite as efficient as properly applied drops, was still very effective.

HITSCHLER, Philadelphia.

FIFTEEN CASES OF CASEOUS PARANASAL SINUSITIS AND A CONSIDERATION OF THE CAUSE OF THIS DISEASE. R. OSAWA and Y. NAKAMURA, J. Oto-Rhino-Laryng. Soc. Japan **56**:608, 1953.

Osawa and Nakamura treated 15 cases of caseous paranasal sinusitis, of which 4 were of the mild type and the others of the severe type, according to Texier's classification. Six of them were between the ages of 50 and 60 years. Eight had involvement in the maxillary sinus.

Foul rhinorrhea, headache, and granular transformation on the wall of the middle meatus were pronounced in the severe type of this disease. In most cases appearance of the infected sinus in x-ray photographs was dark. Destruction of the inner wall of the maxillary sinus and the septum of ethmoid cells was frequently found. The differentiation between carcinoma of the maxillary sinus and this type of suppurative paranasal sinusitis is stressed. Osawa and Nakamura pointed out a few important criteria for differential diagnosis.

From the history and the clinical, pathological, and bacteriological findings, the following clues to the etiology of caseous paranasal sinusitis were mentioned. The mild type of this disease should be considered an atypical form of chronic paranasal sinusitis. The acute, progressive, and destructive nature of the disease and the presence of mixed infection found in severe cases support the view that it may be different from the mild type and may be caused by some bacteria. In four cases of the severe type, abundant spirochetes and fusiform bacteria were found. Analysis of the caseous substance showed the content of whole cholesterol to be as high as 0.39%. Osawa and Nakamura consider these important factors in the development of the severe type of caseous paranasal sinusitis and destruction of bone tissue.

HARA, Los Angeles.

Miscellaneous

THYMUS PROBLEM. J. T. LITTLETON, D. S. MOTSAY, and S. P. PERRY, A. M. A. Am. J. Dis. Child. **86**:705 (Dec.) 1953.

Littleton and his associates state that "the thymus has been maligned." Three years ago at the Guthrie Clinic and Robert Packer Hospital they undertook a review of the thymus problem under a grant-in-aid from the Edwards Memorial Fund. Some 2,600 infants were completely studied clinically and by x-ray and laboratory investigations. Five main theories have been formulated concerning how the thymus produces symptoms and death: (1) mechanical pressure on the trachea—the obstructive theory; (2) the theory of status thymicolymphaticus, advanced by Poultouf (1889) and supported by Warthin and others, which ascribes that death is due to a constitutional defect associated with general hyperplasia of the adrenals and cardiovascular system; (3) the theory of anaphylaxis; (4) the theory that death is due to excessive and abnormal thymic secretion, resulting in lymphotoxemia, and (5) the theory that death is due to hypersusceptibility to some physical or chemical agent. Chevalier Jackson is reported to have observed tracheal compression in 300 bronchoscopies in infants. "This work has not been repeated." The Medical Research Council with the Pathological Society of Great Britain and Ireland, as reported by Young and Turnbull in 1931, states that "no evidence that so-called 'status thymico-lymphaticus' has any existence as a pathological entity." Littleton and his associates state in summary that there is little concrete evidence to support any of the five theories, as mentioned above, linking the thymus with cough, stridor, and cyanosis or with sudden death. These symptoms are still being treated by radiologists at the request of pediatricians and other specialists in cases in which there is a widened mediastinal shadow. The three researchers believe that a definite clinical entity of low-grade, chronic, or recurrent tracheobronchitis has been overlooked.

I. W. VOORHEES, New York.

SPEECH EXAMINATION OF A GROUP OF INSTITUTIONALIZED MENTALLY HANDICAPPED CHILDREN. BERNARD B. SCHLANGER, J. Speech & Hearing Disorders **18**:339 (Dec.) 1953.

Seventy-four mentally retarded children enrolled in St. Coletta's School for Exceptional Children studied for articulatory proficiency showed 30% hearing losses, marked deficiency in auditory memory span for vowels, and sound discrimination. Syllable and tapping rates were slow. In general, their speech was immature. Scores on formal testing with picture stimuli differed from conversation scores. Interrelationships between the five measures presumed to affect articulatory proficiency indicated that these measures were not related to a highly significant degree, with the exception of the maximum repetitive rates for syllable production and tapping.

PALMER, Wichita, Kan.

ANÆSTHETIC PROBLEMS IN OTO-RHINO-LARYNGOLOGY. JOHN GILLIES, J. Laryng. & Otol. **67**:78 (Feb.) 1953.

Anesthesia today need no longer be considered a "necessary evil" but, instead, may even play a supportive role in the care of a patient. Modern methods of anesthesia employ specific agents to effect marked but nontoxic physiological derangement. These agents are capable of producing abolition of muscle tone, apnea, and hypotension under controlled conditions. Their essential merit is reversibility.

The problems encountered are not new ones. Intubation, except in infants, is the safest and most effective solution of the problem of ventilation control and protection of the air passages. The hazard of explosion is greatly diminished by excluding cyclopropane or any of the ethers when diathermy is being used or endoscopy is in progress. Local analgesia is probably the safest method for any endoscopic work, although it is not completely without danger. Deaths following spraying of the air passages are probably related not to the total quantity of analgesic applied but, instead, to its concentration in the patient's blood and the speed at which this is built up. The mucosa of the distal part of the respiratory tract has an increased vascularity, and because of this, absorption here may be almost as rapid as intravenous injection. The advantageous use of muscle relaxants in endoscopy has been established. The essential danger attending curarization during such procedures is the jeopardizing of pulmonary ventilation. The availability of oxygen delivery deep into the air passages and the opportunity to check for cyanosis minimize this hazard.

Troublesome bleeding is a serious handicap for otolaryngologists. This problem can be largely overcome by the use of regional analgesia and the caution of muscle relaxants. The other merits of local anesthesia are, of course, not to be overlooked. During surgical operations under general anesthesia the regular factors which may increase bleeding are vasodilatation, increased pulse pressure, and active circulatory reflexes. These are all interrelated, vary with the plane of anesthesia, but occur regardless of the anesthetic agent used. The mechanisms with chloroform, ether, cyclopropane and thiopental are discussed. Excessive bleeding may also be prevented by induced hypotension. The three most commonly used methods of accomplishing this are discussed. They are (1) reduction of blood volume by arteriotomy, (2) total sympathetic paralysis by the injection of an analgesic drug into the subarachnoid space or into the extradural space, and (3) a generalized ganglionic blockade by the intravenous injection of certain compounds of methonium. Planned hypotension is contraindicated in certain organic disease. When this method is used, the possibility of reactionary hemorrhage must be constantly borne in mind by the surgeon during the course of the operation.

LEJEUNE and NORMANN, New Orleans.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Third International Congress of Bronchoesophagology.
Place: Lisbon, Portugal. Time: September or October, 1954.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: United States of America. Time: 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary: Dr. Hugh A. R. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Walter H. Theobald, 307 N. Michigan Blvd., Chicago 1.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston.

American Broncho-Esophagological Association

President: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston.

American Laryngological Association

President: Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Boston.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
President-Elect: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston.

SECTIONS:

Eastern.—Chairman: Dr. Daniel S. Cunning, 118 E. 53rd. St., N. Y. 22.
Place: Hotel Syracuse, Syracuse, N. Y.
Southern.—Chairman: Dr. William C. Wolfe, 332 W. Broadway, Louisville 2.
Place: Andrew Jackson Hotel, Nashville, Tenn.
Middle.—Chairman: Dr. French K. Hansel, 634 N. Grand Blvd., St. Louis 3.
Place: The Drake, Chicago.
Western.—Chairman: Dr. Guy L. Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Place: The Elks Club, Los Angeles.

American Otological Society, Inc.

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Statler Hotel, Boston.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Harry Neivert, 555 Park Ave., New York 21.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
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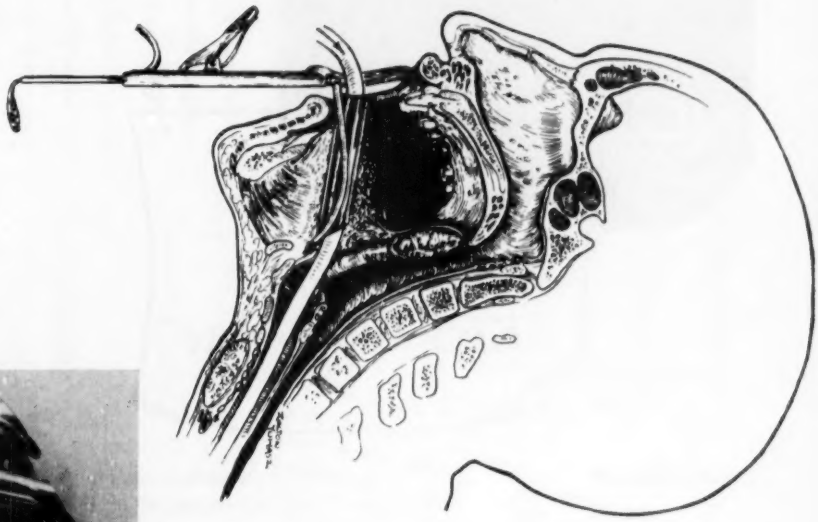

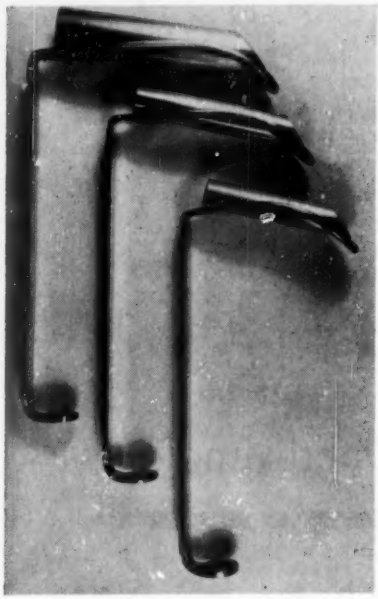

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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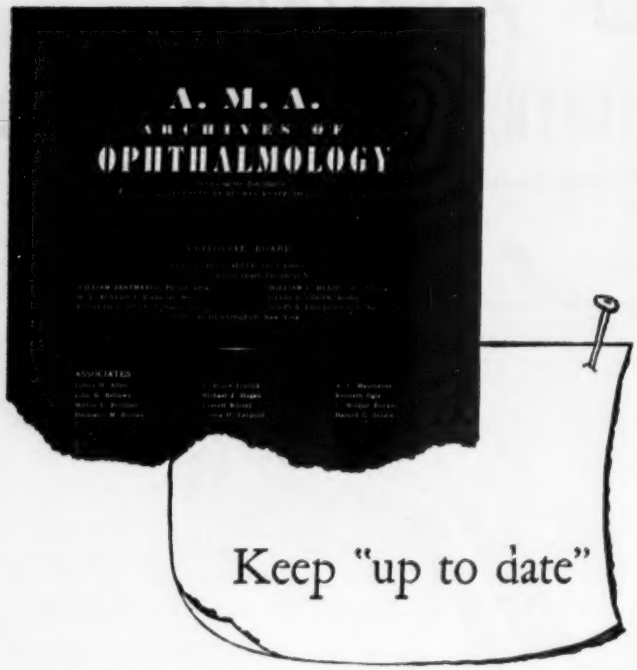

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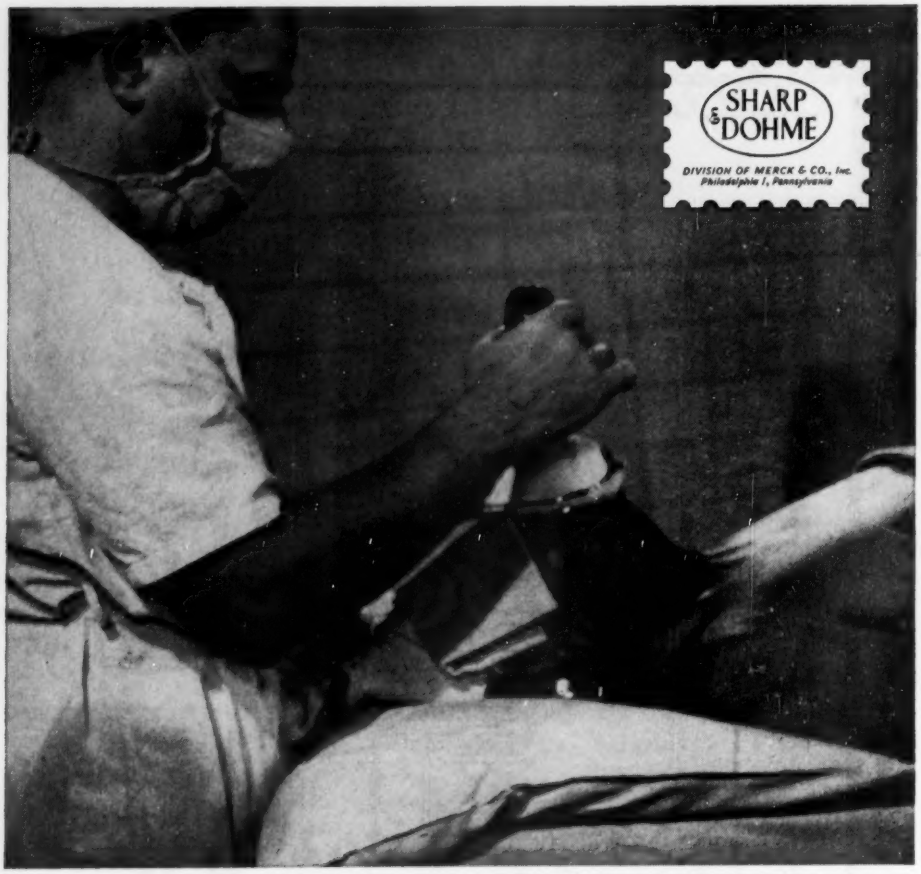

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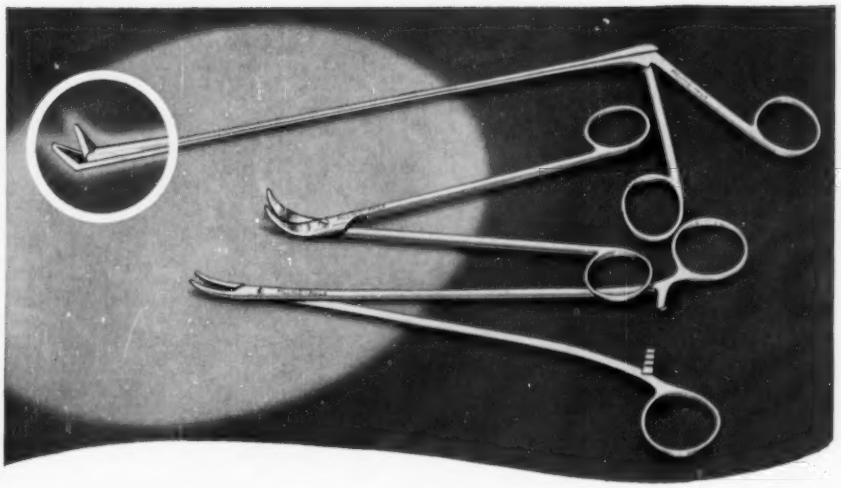

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